

Philipps



Universität
Marburg

FEAR-AVOIDANCE BELIEFS, COPING UND BIOFEEDBACK

Was wird bei der Entstehung und in der Behandlung chronischer Rückenschmerzen
gelernt?

Dissertation

zur Erlangung des Doktorgrades der Naturwissenschaften
(Dr. rer. nat.)

dem Fachbereich Psychologie der Philipps-Universität Marburg
vorgelegt von

Robert Sielski
aus Świecie

Marburg an der Lahn, Dezember 2016

Vom Fachbereich Psychologie der Philipps-Universität Marburg (Hochschulkennziffer 1080) am
30.01.2017 als Dissertation angenommen.

Erstgutachterin: **Dr. Julia A. Glombiewski**

Zweitgutachter: **Prof. Dr. Winfried Rief**

Tag der mündlichen Prüfung: 30.01.2017

DANKSAGUNG

[Die Danksagung ist nicht Teil der Veröffentlichung]

Inhaltsverzeichnis

1	Zusammenfassung und Abstract	1
1.1	Zusammenfassung.....	1
1.2	Abstract	3
2	Theoretischer Hintergrund	5
2.1	Relevanz akuter und chronischer Rückenschmerzen.....	5
2.2	Entstehung und Aufrechterhaltung chronischer Rückenschmerzen aus lerntheoretischer Perspektive.....	7
2.2.1	Fear-Avoidance Modell (FAM)	7
2.2.2	Klassisches Konditionieren als Lernmechanismus im FAM	10
2.2.3	Operantes Konditionieren als Lernmechanismus im FAM	11
2.2.4	Evaluatives Konditionieren als Lernmechanismus im Erwerb von Einstellungen	12
2.3	Lernen in der Behandlung chronischer Rückenschmerzen	15
2.3.1	Biofeedback in der Behandlung chronischer Rückenschmerzen	16
2.3.2	Graduierte Exposition in der Behandlung chronischer Rückenschmerzen	18
3	Darstellung des Dissertationsvorhabens	20
3.1	Relevanz und Herleitung der Fragestellungen	20
3.2	Zielsetzung und Fragestellungen des Dissertationsvorhabens	22
4	Zusammenfassung der Studien	23
4.1	Studie 1: Die Rolle von evaluativem Konditionieren in der Entstehung von Fear-Avoidance Beliefs – experimentelle Studien zur Überprüfung impliziter und expliziter Einstellungen gegenüber rückenbeanspruchenden Bewegungen.	23
4.2	Studie 2: Entwicklung und psychometrische Überprüfung einer deutschen Version des Pain Solutions Questionnaire (PaSol) zur Erfassung assimilativer und akkommodativer Copingstrategien bei chronischen Schmerzen.	27
4.3	Studie 3: Die Wirksamkeit von Biofeedback in der Behandlung chronischer Rückenschmerzen	30
4.4	Studie 4: Biofeedback als psychologisches Behandlungsverfahren für chronische Rückenschmerzen.	33
5	Zusammenfassende Diskussion und Ausblick	35
5.1	Einschränkungen	36
5.2	Perspektiven für die Forschung und Implikationen für die klinische Praxis	38
5.3	Fazit	41
	Literaturverzeichnis	42

Appendix.....	50
A: Studien	50
A.1 Studie 1.....	50
A.2 Studie 2.....	90
A.3 Studie 3.....	113
A.4 Studie 4.....	130
B: Tabellarischer Lebenslauf und Publikationen	134
C: Eidesstattliche Erklärung.....	136

Abbildungsverzeichnis

Abb. 1: Fear-Avoidance Modell (nach Vlaeyen und Linton, 2000).....	8
Abb. 2: Klassische und operante Konditionierung von Fear-Avoidance (nach Vlaeyen und Linton, 2000).	12

„Alle Einführungswerke zur Verhaltenstherapie stellen die Lerntheorien an den Beginn der Abhandlung. Das hat seinen Hauptgrund darin, dass in den Grundlagen Merkmale des Lernens sowohl für die Entstehung als auch für die Therapie von psychischen Störungen als zentral angesehen werden.“ (Reinecker, 2005, S. 75)

1 ZUSAMMENFASSUNG UND ABSTRACT

1.1 Zusammenfassung

Das einleitende Zitat von Reinecker betont die Relevanz lerntheoretischer Annahmen in der Entwicklung, Aufrechterhaltung sowie Behandlung psychischer Störungen. Im Bereich chronischer Rückenschmerzen wird häufig das Fear-Avoidance Modell nach Vlaeyen und Linton (2000) herangezogen, um die Entstehung und Aufrechterhaltung chronischer Rückenschmerzen zu erklären. Laut Modell werden bei der Entwicklung klassische und bei der Aufrechterhaltung operante Konditionierungsprozesse angenommen. Diese Lernmechanismen können jedoch nicht vollständig den Teufelskreis aus Schmerz, Angst und Vermeidung erklären. Dies gilt insbesondere für die Entstehung der kognitiven Komponenten des Modells wie den Fear-Avoidance Beliefs.

Für die Behandlung chronischer Rückenschmerzen liegen mittlerweile zahlreiche Interventionen vor, die mit verschiedenen Methoden unterschiedliche Behandlungsziele (z.B. Schmerzreduktion vs. Lebensqualität trotz Schmerzen) verfolgen, sodass Patienten andere Copingstrategien lernen. Erste Befunde geben Hinweise, dass in der Bewältigung chronischer Schmerzen akkommodative Copingstrategien hilfreicher als assimilative Copingstrategien sind. Sowohl für die Untersuchung der eingesetzten Copingstrategien als auch die Auswirkungen derer auf die Symptomatik sind adäquate Messinstrumente essentiell.

Für Schmerzerkrankungen wie Fibromyalgie, Migräne und Kopfschmerz vom Spannungstyp konnte Biofeedback als wirksames psychologisches Verfahren belegt werden. Im Bereich chronischer Rückenschmerzen ist die Befundlage unklar und bedarf einer genaueren Überprüfung der kurz- und langfristigen Wirksamkeit.

Die Entstehungsmechanismen von Fear-Avoidance Beliefs wurden trotz der Relevanz in der Entwicklung und Aufrechterhaltung chronischer Rückenschmerzen bislang wenig untersucht. Aus diesem Grund wurden in Studie 1 der vorliegenden publikationsbasierten Dissertation untersucht, ob evaluatives Konditionieren – als weiterer Konditionierungsprozess – Einstellungen gegenüber rückenbeanspruchenden Bewegungen im Sinne der Fear-Avoidance Beliefs verändern kann. In zwei durchgeführten Experimenten konnte gezeigt werden, dass durch evaluatives Konditionieren

negative aber nicht positive Einstellungen gegenüber rückenbeanspruchenden Bewegungen gebildet wurden. Negative Veränderungen konnten sowohl auf expliziter als auch teilweise auf impliziter Ebene gezeigt werden. Die Ergebnisse legen nahe, dass evaluatives Konditionieren ein relevanter Lernmechanismus in der Entwicklung von Fear-Avoidance Beliefs ist.

In Studie 2 wurde in einer Stichprobe von $N = 165$ Patienten mit chronischen Rückenschmerzen eine deutsche Version des Pain Solutions Questionnaires (PaSol) entwickelt und auf deren Gütekriterien untersucht. Die deutsche Version des PaSol zeigte gute psychometrische Qualitäten. Die Überprüfung der Faktorstruktur ergab dieselbe Item-Skalenzuordnung und vier zugrunde liegende Faktoren wie in der Originalversion des Fragebogens berichtet. Zusätzlich konnten erste Hinweise gegeben werden, dass der PaSol sensitiv ist, um Veränderungen durch Psychotherapie aufzuzeigen. Mit der Entwicklung einer reliablen und validen Version des PaSol liegt nun auch für den deutschen Sprachraum ein adäquates Messinstrument vor, welches den Einsatz von Copingstrategien und somit den Umgang mit Schmerzen differenziert erfassen kann.

Im Rahmen einer Metaanalyse wurde in Studie 3 die Wirksamkeit der Behandlung chronischer Rückenschmerzen mit Biofeedback untersucht. Es wurden 21 Studien in die Analysen aufgenommen, die einen Biofeedbackanteil von mindestens 25% an der Gesamtbehandlungszeit berichteten und mindestens eine der folgenden Zielgrößen untersuchten: Schmerzintensität, Depression, Beeinträchtigung, Selbstwirksamkeitserwartungen oder muskuläre Anspannung. Die Ergebnisse deuten auf kleine bis mittlere Wirksamkeit für alle genannten Zielgrößen, die im Vergleich zu Kontrollgruppen als auch nach einem Follow-Up von durchschnittlich acht Monaten stabil und vergleichbar blieben. In Studie 4 wurden die Ergebnisse der Metaanalyse vertiefend diskutiert und auf dessen Wirkmechanismen sowie Limitationen eingegangen. Insgesamt konnte Biofeedback als wirksame (zusätzliche) psychologische Intervention in der Behandlung chronischer Rückenschmerzen nachgewiesen werden. Die Ergebnisse sprechen gleichzeitig jedoch auch dafür weitere Interventionen einzusetzen, insbesondere wenn es sich um eine stark beeinträchtigte Patientengruppe handelt.

1.2 Abstract

Learning is crucial for the development, maintenance, and treatment of mental disorders. The fear-avoidance model (Vlaeyen & Linton, 2000) is frequently used to explain the development and maintenance of chronic back pain through classical and operant conditioning procedures. However, inconsistent evidence about the presence of a conditioned fear response suggests further learning mechanisms to be important; especially in the formation of fear-avoidance beliefs.

Cognitive-behavioral therapy offers a wide range of interventions in the treatment of chronic back pain. Depending on the intervention, the used methods and targeted therapy goals might differ, from reducing pain intensity to improving quality of life despite being in pain. First studies suggest that accommodative coping strategies might be more beneficial in the treatment of chronic pain compared to assimilative coping strategies. Adequate measurements are essential for investigating the used coping strategies as well as the consequences of their utilization on pain-related symptoms.

Biofeedback has been found to be an effective psychological intervention in the rehabilitation of fibromyalgia, migraine, and tension-type headache. However, the empirical evidence in the treatment of chronic back pain shows inconsistent results and remains unclear.

In this thesis, four Studies are presented that investigate said aspects.

Despite their relevance in the development and maintenance of chronic back pain, there is little knowledge on the mechanisms behind the acquisition of fear-avoidance beliefs. On that account, in Study 1, we aimed at examining whether evaluative conditioning affected attitudes toward back-stressing movements as found in fear-avoidance beliefs. Results from two experiments showed that evaluative conditioning formed negative (but not positive) attitudes toward back-stressing movements on an explicit level and partially on an implicit level. Thus, our results indicate that evaluative conditioning might play a role in the acquisition of fear-avoidance beliefs.

In Study 2, we developed a German version of the Pain Solutions Questionnaire (PaSol) and analyzed its psychometric properties in a sample of $N = 165$ patients suffering from chronic back pain. The exploratory factor analysis reproduced the original questionnaire's four-factor structure. The reliability and validity analyses demonstrated acceptable to good results. Moreover, the PaSol was found to be sensitive to detect treatment changes over time. The German version of the PaSol is a reliable and valid instrument in the measurement of assimilative and accommodative coping strategies in patients suffering from chronic low back pain.

In Study 3, we investigated the efficacy of biofeedback as treatment option for chronic back pain by conducting a meta-analysis based on 21 studies that employed a biofeedback intervention for at least 25 % of the total treatment time and reported at least one of the following outcomes: pain intensity, depression, disability, self-efficacy, and reduction of muscle tension. Effect size estimates for the total sample suggested a small to moderate effect for all outcomes. These effects remained comparatively stable over an average follow-up phase of 8 months and for controlled studies only. Thus, we conclude that biofeedback can be beneficial for various pain-related outcomes in the short and long terms. A further discussion on biofeedback as a psychological intervention, both as standalone and as adjunctive intervention for chronic back pain is shown in Study 4. Altogether, our results indicate that biofeedback can lead to improvements on several pain-related outcomes. However, our results also suggest the utilization of further interventions for highly disabled patients.

2 THEORETISCHER HINTERGRUND

2.1 Relevanz akuter und chronischer Rückenschmerzen

Rückenschmerzen werden weltweit zu den am häufigsten vorkommenden Erkrankungen gezählt. Die Wahrscheinlichkeit mindestens einmal im Leben unter akuten Rückenschmerzen zu leiden, wird auf über 80% geschätzt (Balagué, Mannion, Pellisé, & Cedraschi, 2012; Louw, Morris, & Grimmer-Somers, 2007). Dazu gehören mehrheitlich unspezifische Rückenschmerzen, also jene, bei denen keine klare pathologische Ursache wie z.B. eine Entzündung oder Osteoporose vorliegt. Bewegungsmangel oder Schonverhalten werden oft als Ursache für die Schwächung oder Fehlbeanspruchungen der Muskulatur gesehen, die sich langfristig negativ auf die Gesundheit des Rückens auswirkt, jedoch nicht unbedingt zu einer Chronifizierung führen muss (Pfungsten & Hildebrandt, 2011). Das Vorkommen unspezifischer Rückenschmerzen ist unabhängig vom Alter und wird sowohl von Jugendlichen als auch von älteren Personen berichtet (Balagué et al., 2012; Pellisé et al., 2009). Ebenfalls variiert die Inanspruchnahme von medizinischen Behandlungen stark und führt auch bei Betroffenen, die keine ärztliche Hilfe aufsuchen, in der Mehrzahl der Fälle zu einer Genesung und Schmerzfreiheit.

Nichtsdestotrotz kommt es bei jedem fünften Betroffenen zu einer Chronifizierung der Rückenschmerzen (Breivik, Collett, Ventafridda, Cohen, & Gallacher, 2006), die mit persönlichen sowie gesellschaftlichen Kosten verbunden ist. Auf persönlicher Ebene zeigen Personen mit chronischen Rückenschmerzen starke Beeinträchtigung in der Bewältigung ihres Alltags sowie ihrer Arbeitsfähigkeit, erhöhte komorbide Erkrankungen wie Depressionen oder Angststörungen und eine allgemein geringere Lebensqualität. Häufige Arbeitsausfälle, die Beanspruchung von (frühzeitigen) Erwerbs- und Berufsunfähigkeitsrenten sowie die hohe Inanspruchnahme von medizinischen Behandlungen führen zudem zu hohen direkten und indirekten sozioökonomischen Kosten (Gore, Sadosky, Stacey, Tai, & Leslie, 2012; Wenig, Schmidt, Kohlmann, & Schweikert, 2009).

Die anfangs angenommenen biologischen Erklärungsmodelle zum Übergang von akuten zu chronischen Rückenschmerzen konnten die Entstehung sowie Aufrechterhaltung chronischer Rückenschmerzen nicht hinreichend erklären. Neben der unklaren Pathologie für das Auftreten von unspezifischen Rückenschmerzen konnte zudem gezeigt werden, dass auch bei schmerzfreien Personen medizinische Untersuchungen regelmäßig Auffälligkeiten in der Wirbelsäule gefunden werden (Boos et al., 1995; Jensen et al., 1994), sodass sich der Fokus von medizinischen zusätzlich auf psychologische Faktoren erweitert hat. In einer Metaanalyse von Linton (2000) wurden 37 prospektive Studien zu psychologischen Risikofaktoren untersucht und ergeben, dass emotionale,

kognitive sowie behaviorale Faktoren einen bedeutsamen Einfluss sowohl auf das Auftreten chronischer Rückenschmerzen als auch auf den Übergang von akuten zu chronischen Schmerzen hatten. Weitere Studien zeigten, dass unter anderem Angst vor Schmerzen, katastrophisierende Gedanken oder ein dysfunktionaler Umgang mit den Schmerzen das Risiko einer Chronifizierung erhöhen und konnten diese metaanalytischen Befunde stützen (Chou & Shekelle, 2010; Crombez, Vlaeyen, Heuts, & Lysens, 1999; Hasenbring, Marienfeld, Kuhlendahl, & Soyka, 1994; T Pincus, Burton, Vogel, & Field, 2002).

Psychologische Erklärungsmodelle versuchen mithilfe von lerntheoretischen Annahmen sowohl das Auftreten von bestimmten Risikofaktoren in Personen mit akuten Schmerzen als auch die Mechanismen, die dem Übergang von akuten zu chronischen Schmerzen unterliegen, zu erklären. Basierend auf den angenommenen Lernmechanismen wurden im Laufe der Zeit verschiedene Therapieansätze entwickelt, um die Behandlung chronischer Rückenschmerzen zu verbessern. Die vorliegende Arbeit beschäftigt sich mit spezifischen Lernmechanismen, die sowohl in der Entstehung und Aufrechterhaltung als auch in der Behandlung chronischer Rückenschmerzen angenommen werden und dadurch mögliche Veränderungsprozesse einleiten. Neben der Überprüfung etablierter Ansätze in der Behandlung sollen zudem – für die Schmerzforschung – neue lerntheoretische Ansätze untersucht werden.

2.2 Entstehung und Aufrechterhaltung chronischer Rückenschmerzen aus lerntheoretischer Perspektive

In diesem Kapitel soll zunächst auf das Fear-Avoidance Modell (FAM) zur Chronifizierung von Rückenschmerzen eingegangen werden, mit dem versucht wird, die Entstehung und Aufrechterhaltung chronischer Rückenschmerzen aus biopsychosozialer Perspektive zu erklären. Anschließend sollen die dem FAM angenommenen, zugrunde liegenden Lernmechanismen der klassischen und operanten Konditionierung näher betrachtet werden. Abschließend wird ein, in der Schmerzforschung bislang wenig beachteter, Lernmechanismus, nämlich das Evaluative Konditionieren, und dessen mögliche Relevanz für die Entstehung und Aufrechterhaltung chronischer Rückenschmerzen beschrieben.

2.2.1 Fear-Avoidance Modell (FAM)

In der gegenwärtigen Forschung zu chronischen Rückenschmerzen gilt das Fear-Avoidance Modell nach Vlaeyen und Linton (2000) als etabliertes biopsychosoziales Erklärungsmodell für die Entstehung sowie Aufrechterhaltung chronischer Rückenschmerzen (siehe Abbildung 1), das insbesondere durch ein Review dieser Forscher (Vlaeyen & Linton, 2000) an Bedeutsamkeit gewonnen und das Gros der darauffolgenden Schmerzforschung angeregt hat.

Das FAM beschreibt zwei gegensätzliche Möglichkeiten wie auf eine Situation, in der eine Verletzung zu akuten Schmerzen führt, reagiert werden kann: Konfrontation und Vermeidung. Konfrontation beschreibt dabei den Weg der Genesung, Vermeidung hingegen den Weg der Chronifizierung. Wie bereits in Kapitel 2.1 beschrieben, ist die Wahrscheinlichkeit unter akuten Rückenschmerzen zu leiden deutlich höher als chronische Rückenschmerzen zu entwickeln. Deshalb gehen Asmundson und Kollegen (2004) davon aus, dass der Großteil der Betroffenen funktional, d.h. mit Konfrontation, auf akute Schmerzepisoden reagiert, indem sie nach und nach ihre Alltagsaktivitäten wieder aufnehmen und ebenfalls jene Situationen konfrontieren, in denen der Schmerz initial auftrat. Langfristig soll es dadurch zu einem Rückgang der Schmerzen und zur Genesung kommen. Dieser Reaktion geht voraus, dass die Schmerzen und die damit assoziierten Situationen nur als unangenehm wahrgenommen, jedoch nicht katastrophisierend interpretiert werden und somit keine Angst auslösen.

Die Bewertung der Schmerzen als etwas Bedrohliches löst hingegen (schmerzbezogene) Angst aus und führt zu Vermeidung von Situationen, in denen die Schmerzen (wieder-)erlebt oder verstärkt werden könnten. Die Angst vor Schmerzen führt kognitiv zu einer verstärkten Beschäftigung mit schmerzbezogenen Informationen und Hypervigilanz auf potentiell bedrohliche Reize. Aus dem vermehrten Vermeidungsverhalten ergibt sich ein Rückzug aus sozialen, sportlichen sowie

beruflichen Aktivitäten und einer subjektiv wahrgenommenen Beeinträchtigung. Die dadurch entstehende Inaktivität und der Wegfall wichtiger positiver Verstärker können physiologisch wiederum zum Abbau der Muskulatur sowie einer Verschlechterung des allgemeinen Gesundheitszustands, und psychisch zu einer depressiven Symptomatik führen (Verbunt, Seelen, Vlaeyen, Van der Heijden, & Knottnerus, 2003). Sowohl der körperliche Abbau – auch *disuse* genannt – als auch die psychischen Folgen werden mit einer verringerten Schmerztoleranz assoziiert und können bei einer Konfrontation mit einer als bedrohlich eingestuften Situation zu stärkeren Schmerzen führen. Dies wiederum verstärkt die Angst vor Schmerzen bzw. Angst vor Bewegung und fördert die Aufrechterhaltung der Schmerzsymptomatik.

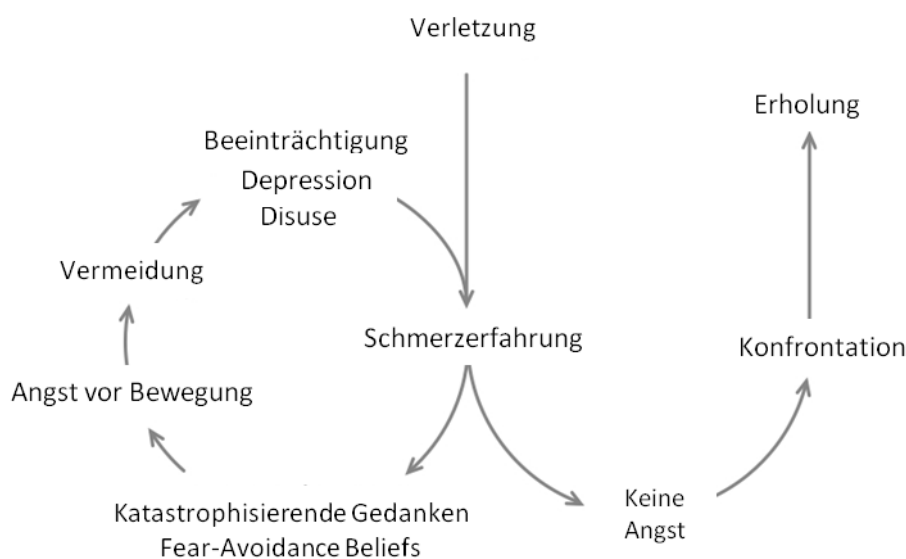


Abb. 1: Fear-Avoidance Modell (nach Vlaeyen und Linton, 2000).

Der biopsychosoziale Ansatz des FAM unterscheidet sich von traditionellen biologisch-medizinischen Erklärungsmodellen insbesondere durch die geringere Bedeutsamkeit der physiologischen Komponente des Schmerzreizes und der Zuschreibung psychologischer Faktoren für die Entwicklung und Aufrechterhaltung chronischer Schmerzen. Studien konnten zeigen, dass schmerzbezogene Angst, katastrophisierende Bewertungen von Schmerzen und negative Überzeugungen gegenüber rückenbeanspruchenden Aktivitäten langfristig mit mehr Beeinträchtigung sowie höherer Schmerzintensität einhergehen (Brox, 2014; Grotle, Vøllestad, & Brox, 2006; Linton, 2000; Woby, Watson, Roach, & Urmston, 2004). Schon früh konnten auch Zusammenhänge zwischen einem passiven Umgangs bzw. Coping mit Schmerzen und Gefühlen von Hilflosigkeit und katastrophisierenden Gedanken gefunden werden (Rosenstiel & Keefe, 1983). Crombez und Kollegen (1999) schlussfolgerten aus Ergebnissen von drei unabhängigen

Studien, dass schmerzbezogene Angst sogar stärker mit subjektiver Beeinträchtigung zusammenhängt als die empfundene Schmerzintensität. Die stärkere Relevanz psychosozialer Faktoren im Vergleich zu physiologischen Komponenten wie Schmerzintensität konnte jedoch nicht konsistent in allen Studien gezeigt werden. In einer längsschnittlich angelegten Untersuchung von Sieben und Kollegen (2005) sagten frühere Erfahrungen mit akuten Rückenschmerzen sowie Schmerzintensität den Verlauf von akuten zu chronischen Rückenschmerzen vorher. Auf psychologischer Ebene war lediglich negativer Affekt ein signifikanter Prädiktor. Obwohl der Anteil biologischer und psychologischer Faktoren für die Entstehung akuter Rückenschmerzen sowie den Übergang von akuten zu chronischen Rückenschmerzen und deren Aufrechterhaltung nicht genau spezifiziert werden kann, ist die Rolle psychologischer Faktoren wie sie im FAM beschrieben ist, unbestritten.

Eine zentrale Rolle im FAM wird der Angst vor Schmerzen oder Angst vor Bewegung (Kinesiophobie) zugeschrieben, auf die mit Vermeidung von Bewegungen reagiert wird, bei denen ein Schmerzerleben erwartet wird, und somit der beschriebene Teufelskreis aufrechterhalten wird (Glombiewski et al., 2015; Meulders, Vansteenwegen, & Vlaeyen, 2011). Weiterhin stehen sogenannte Fear-Avoidance Beliefs (FAB) im Fokus, mit denen Einstellungen, Überzeugungen und Annahmen über rückenbeanspruchenden Aktivitäten oder schmerzspezifische Situationen gemeint sind. FAB kommen nicht nur bei Patienten mit chronischen Rückenschmerzen vor, sondern sind auch in gesunden Populationen verbreitet und werden als Risikofaktor für eine verzögerte Genesung und für Chronifizierung gezählt (Goubert, Crombez, Hermans, & Vanderstraeten, 2003; Grotle, Vøllestad, Veierød, & Brox, 2004; Wertli, Rasmussen-Barr, Weiser, Bachmann, & Brunner, 2014; Woby et al., 2004). FAB sind jedoch nicht unabhängig von schmerzbezogener Angst. Vielmehr konnten Studien auch zeigen, dass diese kognitive Komponente Angst auslösen kann (Goubert, Crombez, & De Bourdeaudhuij, 2004; Leeuw et al., 2007). In der Literatur werden sowohl die emotionale als auch die kognitive Komponente des FAM nicht selten als Fear-Avoidance zusammengefasst und teilweise synonym verwendet oder als ein kognitiv-emotionaler Faktor beschrieben (Goubert, Crombez, & Peters, 2004). Trotz der uneinheitlichen Definition des Fear-Avoidance Begriffs ist in den letzten Jahren ein starker Fokus der Behandlungsansätze bei chronischen Rückenschmerzen auf die Angstreduktion gelegt worden (Leeuw et al., 2008; Vlaeyen, De Jong, Geilen, Heuts, & Van Breukelen, 2001). Pincus und Kollegen (2010) empfehlen in ihrer Übersichtsarbeit zur Evidenz des FAM, den Begriff der Fear-Avoidance einheitlicher zu untersuchen und Patienten in Subgruppen zu unterteilen und Behandlungsansätze entsprechend der Eigenschaften der Patienten anzupassen. Dafür spricht ebenfalls eine fMRT-Studie von Barke und Kollegen (2012), die keine neurologischen

Unterschiede zwischen gesunden Probanden und Patienten mit einer geringen oder hohen Ausprägung von Angst vor Bewegung finden konnten. Die Erwartung einer stärkeren Aktivierung angstrelevanter Hirnregionen (z.B. Amygdala oder Substantia nigra) wie sie bei anderen spezifischen Angststörungen zu finden ist (Etkin & Wager, 2007), konnte demnach nicht belegt werden. Eine mögliche Implikation dieser Ergebnisse liegt nicht nur in der Auswahl der Interventionen bei Patienten mit chronischen Rückenschmerzen, sondern ebenfalls in den zugrundeliegenden Lernmechanismen, die für die Entwicklung einer kognitiven Komponente, im Sinne der Fear-Avoidance Beliefs, bei Patienten mit akuten oder chronischen Rückenschmerzen verantwortlich sein könnten.

2.2.2 Klassisches Konditionieren als Lernmechanismus im FAM

Die Erklärung zur Entstehung und Aufrechterhaltung chronischer Rückenschmerzen wie sie im FAM beschrieben wird, basiert auf zwei Lernmechanismen, die im Zusammenspiel den Teufelskreis aus Schmerz, Angst und Vermeidung antreiben. Trotz der inkonsistenten Ergebnisse bezüglich der Auswirkungen der Schmerzintensität auf die Beeinträchtigung wie sie im vorangegangenen Kapitel dargestellt wurde, stellt Schmerz für diese Lernmechanismen eine bedeutende Rolle dar. Schmerz ist im akuten Zustand ein Signal für eine Verletzung oder Schädigung und löst eine angeborene Abwehrreaktion aus, die sich in der Aktivierung des sympathischen Nervensystems zeigt und unter anderem mit muskulärer Anspannung sowie Angst einhergeht. Gleichzeitig führt Schmerz zu Verhaltensweisen, deren Ziel es ist, den Schmerz zu minimieren (z.B. durch Sicherheitsverhalten) oder schmerzhaft Situationen zu vermeiden. Somit ist Schmerz nicht nur ein physiologisch unangenehmes Erlebnis, sondern ein starker Motivator, der dazu führt, dass im Alltag gelernt wird, welche Situationen als „sicher“ eingeschätzt werden dürfen und welche Situationen „bedrohlich“ sind (Goubert, Crombez, & Peters, 2004). Folglich werden relevante Signale gelernt und Erwartungen gebildet, die dazu führen sollen, potentiell schmerzauslösende Situationen zu umgehen und dadurch den Organismus zu schützen (den Hollander, de Jong, Volders, Goossens, Smeets, Vlaeyen, 2010).

Übertragen auf das FAM stellt Schmerz, mit dem der Kreislauf beginnt, einen salienten unconditionierten Stimulus (US) dar, der eine unconditionierte Abwehrreaktion (UR) auslöst. Dabei ist die Stärke der Abwehrreaktion individuell unterschiedlich. Studien konnten zeigen, dass eine hohe Neigung zu Katastrophisieren sowie Angstsensitivität die subjektiv empfundene Bedrohung von Schmerzen verstärken und zu einer stärkeren Abwehrreaktion führen (Goubert, Crombez, & Van Damme, 2004; Esteve & Camacho, 2008). Wird der Schmerz mit einer spezifischen Bewegung, z.B. Bücken, in Verbindung gesetzt, kommt es zur Bildung einer

Assoziation zwischen dem Schmerz und der Bewegung, sodass die ursprünglich als neutral bewertete Bewegung keinen neutralen Stimulus (NS), sondern einen konditionierten Stimulus (CS) darstellt. Die konditionierte Bewegung dient nun als Signal für die Antizipation der Schmerzen und führt zur unkonditionierten Abwehrreaktion Angst – der CS sagt folglich das Auftreten des US vorher und löst auch ohne Auftreten des US eine UR und in Folge dessen eine konditionierte Reaktion (CR) aus, die sich in diesem Fall in Vermeidungs- oder Sicherheitsverhalten zeigt. Der CS dient entsprechend als Signal für das erwartete Auftreten des US (Signal- bzw. Erwartungslernen; Rescorla, 1988).

Beispielhaft bedeutet dies, dass eine Person, die während des Hebens einer Wasserkiste Schmerzen empfindet, eine Verbindung zwischen dem Schmerzerlebnis und der entsprechenden Abwehrreaktion sowie der spezifischen Bewegung des Hebens herstellt. Diese Verbindung führt dazu, dass die Person erwartet, bei der nächsten Hebebewegung wieder Schmerzen zu empfinden, sodass allein das Ankündigen dieser zu einer Angstreaktion führt und in Vermeidung der Hebebewegung resultiert, um keine Schmerzen erfahren zu müssen. Dieses Beispiel beschreibt einen klassischen Konditionierungsprozess, der über direkte Erfahrung zu einer Angst-Vermeidungs-Verbindung bzw. dem Lernen von Fear-Avoidance führt. Weitere in der Literatur diskutierte Möglichkeiten Fear-Avoidance zu erwerben, sind Beobachtungs- und Instruktionslernen (den Hollander et al., 2010; Helsen, Goubert, Peters, & Vlaeyen, 2011).

2.2.3 Operantes Konditionieren als Lernmechanismus im FAM

Der Erwerb von Fear-Avoidance, insbesondere im Sinne von schmerzbezogener Angst, wird weitestgehend versucht über klassische Konditionierungsprozesse zu erklären. Die Aufrechterhaltung des Verhaltensmusters aus Angst und Vermeidung folgt hingegen operanten Konditionierungsmechanismen. Im FAM wird dabei das Ausbleiben der erwarteten Schmerzen und der Angst als wichtigster negativer Verstärker (C-/) angesehen, der das Vermeidungsverhalten angstbesetzter Bewegungen oder Situationen (s.o. Heben) aufrechterhält und langfristig auf weitere Situationen erweitert. Zudem werden aufgrund des fortwährenden Vermeidungsverhaltens keine korrigierenden Erfahrungen gesammelt, sodass die Richtigkeit dieses Verhaltens nicht infrage gestellt und veränderungsresistent wird (Vlaeyen, Morley, Linton, Boersma, & de Jong, 2012). Zusätzlich kann offen gezeigtes Schmerzverhalten zu Verständnis und mehr Zuwendung von der Familie führen und somit einen positiven Verstärker darstellen. Andererseits wird gesundheitsförderliches Verhalten und Konfrontation mit angstbesetzten Bewegungen nicht direkt belohnt, sondern kann zunächst tatsächlich – auch aufgrund des disuse-

Syndroms – zu verstärkten Schmerzen und Angst führen und wird deshalb konsequenterweise wieder vermieden (Goubert, Crombez, & Peters, 2004; Sanders, 2002).

Die beschriebenen klassischen und operanten Konditionierungsprozesse sind in Abbildung 2 dargestellt.

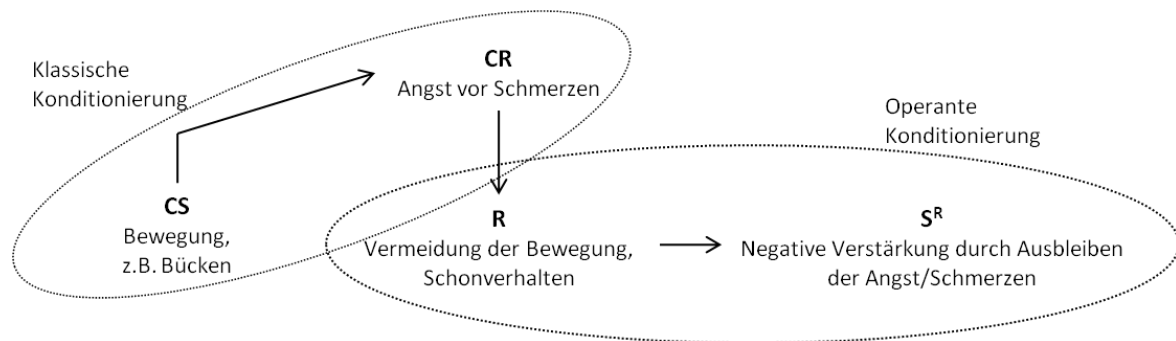


Abb. 2: Klassische und operante Konditionierung von Fear-Avoidance (nach Vlaeyen und Linton, 2000).

Die hier beschriebenen Lernmechanismen basieren mehrheitlich auf der Annahme, dass schmerzbezogene Angst der zentrale Faktor im FAM darstellt und Vermeidungsverhalten auslöst. In der Literatur werden jedoch inkonsistente Befunde gezeigt. Von daher ist anzunehmen, dass auch andere Mechanismen des FAM von Bedeutung sind, insbesondere vor dem Hintergrund, dass Vermeidungsverhalten ebenfalls ohne das Auftreten von Angst gefunden wurde (Goubert, Crombez, & Peters, 2004). Ein Blick auf kognitive Komponenten des FAM in Form von negativen Überzeugungen bzw. Einstellungen gegenüber Schmerzen oder schmerzassoziierten Verhaltensweisen legt nahe, dass Lernmechanismen zur Entwicklung und Veränderung von Einstellungen relevant sein könnten.

2.2.4 Evaluatives Konditionieren als Lernmechanismus im Erwerb von Einstellungen

Literatur zur Entwicklung und Veränderung von Einstellungen, die insbesondere durch Studien aus der Sozialpsychologieforschung besteht, stellt evaluatives Konditionieren als wichtigen Lernmechanismus heraus. Der Begriff evaluatives Konditionieren (EK) wurde von Martin und Levey (1978) geprägt und beschreibt den Transfer der Valenz eines affektiv positiven oder negativen Stimulus auf einen ursprünglich neutralen Stimulus durch die Paarung beider Stimuli. Folglich wird ein ehemals neutraler Stimulus nach der Paarung mit einem affektiv negativen Stimulus negativer bewertet, weil die Valenz des affektiv negativen Stimulus auf den neutralen Stimulus übertragen wird. Ein wichtiger Unterschied zu anderen Konditionierungsparadigmen ist der Fokus auf die Veränderung der Valenz bzw. Bewertung der CS und weniger auf anderen Veränderungen wie dem Hautleitwert (De Houwer, 2007). Die Ähnlichkeit dieser Prozedur zu

klassischem Konditionieren führte dazu, dass auch in Studien zu EK die Terminologie der klassischen Konditionierung (KK) übernommen wurde. Der affektive negative oder positive Stimulus wird hierbei als unkonditionierter Stimulus (US) betrachtet und der nach der Paarung veränderte Stimulus als konditionierter Stimulus (CS). Die Veränderungen können dabei sowohl auf expliziter, bewusster Ebene als auch auf impliziter, unbewusster Ebene passieren und Verhalten dementsprechend bewusst oder unbewusst formen und steuern (Hermans, Crombez, Vansteenwegen, Baeyens, & Eelen, 2002; Jones, Olson, & Fazio, 2010).

Obwohl die Relevanz von EK im Lernen von Einstellungen unbestritten ist, wird die Abgrenzung zum KK diskutiert. Im Gegensatz zu anderen Formen klassischer Konditionierung führt EK hauptsächlich zu Veränderungen in der Bewertung eines Stimulus, demnach wie positiv oder negativ jener Stimulus bewertet wird (Hofmann, De Houwer, Perugini, Baeyens, & Crombez, 2010). Weiterhin wird die CS-US-Verbindung im KK insofern erklärt, als dass der CS als Signal für das erwartete Auftreten des US dient. Im EK wird hingegen diskutiert, inwiefern eine kognitive Repräsentation einer CS-US-Verbindung entwickelt wird, der CS jedoch keinen Prädiktor für das reelle Eintreten des US bedeutet, sondern insbesondere die affektive Beschaffenheit des US annimmt. Hermans und Kollegen (2002; S. 218) beschreiben dies folgendermaßen: "In other words, the CS makes the subject 'think of' the US, without generating an active expectancy of the US". Ein weiterer wichtiger Unterschied zwischen EK und KK sind angenommene Extinktions- bzw. inhibitorische Lernprozesse. Im KK führen nicht verstärkte CS-Präsentationen, z.B. Durchführung einer Bewegung ohne Schmerz, zu einer Schwächung der Signalwirkung des CS. Diese Schwächung wird mittlerweile nicht mehr der Löschung der CS-US-Verbindung zugeschrieben, sondern mit dem Aufbau einer neuen inhibitorischen CS-noUS-Verbindung erklärt (Bouton, 1994). In Studien zu EK wurde hingegen mehrheitlich Resistenz gegenüber eines solchen Extinktionsprozesses gefunden. Die reine Präsentation des CS ohne US führte in den meisten Untersuchungen nicht zu einem Rückgang der Bewertung des CS, sondern blieb weitestgehend stabil (Baeyens, Díaz, & Ruiz, 2005; Houwer, Thomas, & Baeyens, 2001). Gawronski und Kollegen (2014) konnten ferner zeigen, dass insbesondere auf impliziter Ebene die Veränderung der CS-Bewertung resistent gegenüber CS-noUS-Präsentationen sind. Aufgrund dessen wird zur Veränderung von Einstellungen, die über EK entstanden sind, eine Gegenkonditionierung vorgeschlagen, in der der CS mit einem US gepaart wird, dessen Valenz gegensätzlich zur Valenz in der Lernphase ist, ergo negativ statt positiv vice versa (Kerkhof, Vansteenwegen, Baeyens, & Hermans, 2011; Raes & De Raedt, 2012). Eine weitere Möglichkeit ist die US-Revaluation, die die nachträgliche Veränderung der Valenz des US beschreibt, wodurch die Valenz des CS ebenfalls verändert wird (Raes & De Raedt, 2012). In einer Meta-Analyse zu EK fanden Hofmann und

Kollegen (2010), dass EK im Vergleich zu KK nur wenig von der statistischen Kontingenz von CS und US abhängig ist (Vgl. KK: Rescorla, 1966). Dieses Merkmal von EK ist insofern nennenswert, da es in der Realität außerhalb experimenteller Laborbedingungen selten eindeutige Kontingenzen zwischen CS und US gibt und die relative Unabhängigkeit von dieser dazu führt, dass mehr Situationen für das Lernen von Einstellungen infrage kommen (Walther, Nagengast, & Trasselli, 2005).

Im Bezug auf das FAM empfehlen Gheldof und Kollegen (2004) aus verschiedenen Gründen die Untersuchung von negativen schmerzbezogenen oder rückenbeanspruchenden Einstellungen (Fear-Avoidance Beliefs). Einstellungen können sich auf expliziter bewusster Ebene sowie impliziter unbewusster Ebene zeigen und entsprechend automatisch Verhalten steuern oder formen. Ein besseres Verständnis über FAB könnte folglich ambivalentes Verhalten von Patienten erklären, die die negativen Konsequenzen von Passivität und Vermeidung kennen, diese jedoch weiter ausführen. Im klinischen Kontext könnten ambivalente implizite und explizite Einstellungen therapeutische Interventionen behindern bzw. die Behandlungserfolge mindern. Goubert und Kollegen (2003) fanden in einer gesunden Stichprobe negative implizite Einstellungen gegenüber rückenbeanspruchenden Bewegungen. Besonders interessant erscheint dieses Ergebnis vor dem Hintergrund, dass keine Akquisitionsphase im Sinne einer Konditionierung voranging.

Bislang fehlen jedoch Studien über Lernmechanismen, die dem Erwerb von Fear-Avoidance Beliefs unterliegen. Demzufolge konnte die Relevanz von evaluativem Konditionieren als potentielle Konditionierungsart im Bereich der Fear-Avoidance noch nicht geklärt werden. Aufgrund der spezifischen Merkmale von EK – z.B. der Resistenz gegenüber Extinktion – erscheint es sowohl aus theoretischer als auch aus klinisch-praktischer Perspektive sinnvoll, diese Konditionierungsart zu untersuchen.

2.3 Lernen in der Behandlung chronischer Rückenschmerzen

Nachdem nun die lerntheoretischen Hintergründe bei der Entwicklung und Aufrechterhaltung chronischer Rückenschmerzen erläutert wurde, soll im Folgenden auf die Lernmechanismen eingegangen werden, die psychotherapeutischen Behandlungen unterliegen.

Kognitive Verhaltenstherapie (KVT) kann gegenwärtig als eine der wichtigsten psychologischen Behandlungsformen für chronische Schmerzen betrachtet werden (Morley, 2011). Interventionen aus der KVT basieren in der Regel darauf, neue Strategien zum Umgang mit chronischen Schmerzen zu lernen oder dysfunktionale Annahmen, Erwartungen oder Verhaltensweisen, die mit der Symptomatik zusammenhängen umzulernen bzw. neu zu entwickeln (Stavemann, 2008). Die angewandten psychotherapeutischen Techniken und Ziele können sehr variabel sein und haben sich in den letzten Jahrzehnten stetig verändert und erweitert. Entsprechend der angewandten Technik unterliegen den Interventionen andere Lernmechanismen, die zum Ziel einen verbesserten Umgang mit der chronischen Schmerzstörung haben.

Im Zwei-Prozess-Modell von Brandtstädter und Renner (1990) werden zwei gegensätzliche Modi (assimilativ vs. akkommodativ) zum Umgang mit aversiven oder unkontrollierbaren Lebensereignissen, wie zum Beispiel chronischen Erkrankungen, beschrieben. Die ursprünglich auf Entwicklungsregulation durch Veränderungen im Alter bezogene Theorie wurde von Schmitz, Saile und Nilges (1996) auf chronische Schmerzen übertragen und hat auch aufgrund neuer Therapieansätze vermehrt Anwendung in der Erklärung und Behandlung psychischer Störungen gefunden (De Vlieger, Bussche, Eccleston, & Crombez, 2006; Rief et al., 2015).

Laut Zwei-Prozess-Modell können Personen assimilative oder akkommodative Copingstrategien einsetzen, wenn die Verfolgung persönlicher Ziele aufgrund von veränderten Lebensumständen und daraus resultierenden Hindernissen oder Schwierigkeiten, bedroht scheint oder behindert ist. Assimilativ bedeutet in diesem Zusammenhang, hartnäckig an den Zielen festzuhalten und durch aktives Handeln die Hindernisse zu überwinden. Dies kann einerseits mithilfe erhöhter Anstrengung und andererseits durch die Erweiterung eigener zielrelevanter Kompetenzen geschehen. Führen assimilative Copingstrategien jedoch nicht zur erhofften Zielerreichung, wird angenommen, dass akkommodative Copingstrategien eingesetzt werden (sollten). Der Akkommodationsprozess beinhaltet die Flexibilität, sich von nicht erreichbaren, blockierten Zielen zu lösen und neue – an die Lebensumstände angepasste – Ziele zu formulieren. Beide Modi werden als gegensätzlich betrachtet und können sich bei chronischen Erkrankungen, die komplexere Anforderungsmuster vorweisen, ergänzen (Brandtstädter, 2007).

In Bezug auf chronische Schmerzen ist Schmerzfreiheit ein Wunsch, den Betroffene oft hartnäckig verfolgen, und häufig bei Behandlungsbeginn als wichtigstes Ziel formulieren. Im Gegensatz zur Behandlung akuter Schmerzen, in der Schmerzfreiheit ein angestrebtes Ziel ist, sind Behandlungen chronischer Schmerzen weniger erfolgreich hinsichtlich Schmerzreduktion oder Schmerzfreiheit (Turk, 2002). Im Fall chronischer Schmerzen beschreiben Crombez und Kollegen (2008) assimilative Copingstrategien als solche Anstrengungen, die auf eine Heilung der Schmerzsymptomatik gerichtet sind. Mit akkommodativen Copingstrategien hingegen sind jene gemeint, die das Ziel haben, den Fokus von der erhofften Schmerzfreiheit zu lösen und neue Ziele zu setzen, die trotz der Schmerzen erreicht werden können. Rief und Kollegen (2015) beziehen sich im Kontext desselben Modells vielmehr auf Erwartungen, die Patienten im Bezug auf ihre Erkrankung haben können, und beschreiben Akkommodation als Lernen neuer korrigierender Erfahrungen („Das Heben einer Kiste schadet meinem Rücken nicht!“) wohingegen Assimilation diesen förderlichen Prozess unterbindet, indem auf erwartungskongruente Informationen („Während des Hebens habe ich ein Ziehen in meinem Rücken gespürt!“) fokussiert wird. Dieser Fokus bedeutet gleichzeitig, dass an der ursprünglichen Erwartung festgehalten und aktiv gehandelt wird, um diese wahrscheinlicher zu machen („Es muss einen Weg geben wieder schmerzfrei zu werden, also suche ich weiter danach!“) (vgl. auch: Rief & Glombiewski, 2016).

Hinsichtlich chronischer Schmerzen zeigen Studien teilweise, dass ein assimilativer Umgang mit mehr negativen Konsequenzen wie Beeinträchtigung oder katastrophisierenden Gedanken zusammenhängt (Aldrich, Eccleston, & Crombez, 2000; Crombez, Van Damme, & Eccleston, 2005; Van Damme, Crombez, & Eccleston, 2002). Akkommodative Strategien konnten hingegen mit mehr positiven Effekten assoziiert werden (McCracken, Carson, Eccleston, & Keefe, 2004; Wrosch, Scheier, Miller, Schulz, & Carver, 2003).

Insgesamt werden in der KVT sowohl assimilative als auch akkommodative Interventionen durchgeführt. Die angenommenen Copingstrategien sowie Lernmechanismen, die den jeweiligen Behandlungsmethoden unterliegen, sollen im weiteren Abschnitt für zwei Behandlungsformen beschrieben werden.

2.3.1 Biofeedback in der Behandlung chronischer Rückenschmerzen

Biofeedback wurde bereits in den 1970er Jahren als Behandlungselement psychotherapeutischer Interventionen eingeführt und seitdem auch aufgrund der fortschreitenden Technologien weiterentwickelt (z.B. Vielfalt an Modalitäten, physiologischer Parameter oder Art der Rückmeldung) sowie auf verschiedene Störungsbilder appliziert (Morley, 2011; Neblett, 2016). Dabei kann dieses respondente Verfahren als eigenständige oder ergänzende Intervention

eingesetzt werden. Biofeedback ist eine therapeutische Methode, bei der physiologische, oft unbewusst ablaufende Prozesse des autonomen oder zentralen Nervensystems mit auditiven, visuellen oder taktilen Reizen an den Patienten rückgemeldet werden. Durch das Feedback sollen Patienten mehr Bewusstsein und anschließend Kontrolle über körperliche Prozesse, z.B. Muskelanspannung, lernen, um diese dadurch positiv zu beeinflussen (Neblett, 2016; Rief & Birbaumer, 2011). Biofeedback folgt dabei Prinzipien der operanten Konditionierung, indem der Patient versuchen soll mithilfe des Feedbacks sowie durch Unterstützung des Therapeuten die eigenen körperlichen Prozesse selbst zu regulieren (Schwartz & Schwartz, 2003). Ziele der Biofeedbackbehandlung können – durch das Lernen von Einflussnahme auf physiologische Prozesse – unter anderem die Reduktion von Muskelanspannung oder die Erhöhung der Selbstwirksamkeit oder Copingstrategien wie Entspannung sein. Entspannungstechniken konnten als förderlich für die Schmerzreduktion festgestellt werden (Henschke et al., 2010). Somit kann man Biofeedback zu assimilativen Interventionsverfahren zählen, da es darauf abzielt, den Schmerz mittels Entspannung zu reduzieren bzw. zu kontrollieren. Meta-Analytische Untersuchungen konnten die Wirksamkeit von Biofeedback in der Behandlung anderer Schmerzstörungen, nämlich Fibromyalgie und Kopfschmerz vom Spannungstyp sowie Migräne, belegen (Glombiewski, Bernardy, & Häuser, 2013; Nestoriuc & Martin, 2007; Nestoriuc, Rief, & Martin, 2008). In der Behandlung chronischer Rückenschmerzen zeigen sich inkonsistente Befunde zur Wirksamkeit von Biofeedback. In einer Meta-Analyse zu psychologischen Behandlungen bei chronischen Rückenschmerzen konnte zwar gezeigt werden, dass sogenannte „self-regulatory treatments“, zu denen Biofeedback gezählt wird, positive Effekte auf Schmerzintensität und Depression haben, jedoch kann die Wirksamkeit nicht spezifisch auf Biofeedback zurückgeführt werden, da ebenfalls Entspannungstrainings dazu gezählt wurden (Hoffman, Papas, Chatkoff, & Kerns, 2007). Weitere Studien verglichen Behandlungen mit Biofeedbackelementen gegen physiotherapeutische oder KVT-Behandlungen ohne Biofeedback und konnten sowohl kurz- als auch langfristige Überlegenheit in der Wirksamkeit auf Schmerzintensität, Depressivität und physiologische Faktoren für Biofeedback zeigen (Flor & Birbaumer, 1993; Magnusson, Chow, Diamandopoulos, & Pope, 2008). Neben diesen vielversprechenden Befunden stehen ebenfalls Studien, in denen die Ergänzung mit Biofeedback in einer regulären KVT-Behandlung keine zusätzlichen Verbesserungen hervorbringen konnte oder in denen keine Wirksamkeit von Biofeedback auf die Reduktion der Symptomatik bei chronischen Rückenschmerzen gezeigt werden konnte (Glombiewski, Hartwich-Tersek, & Rief, 2010; Nouwen, 1983; Stuckey, Jacobs, & Goldfarb, 1986). Aufgrund dieser inkonsistenten

Befundlage kann abschließend nicht schlussfolgert werden, ob Biofeedback als wirksame Intervention bei chronischen Rückenschmerzen eingesetzt werden sollte.

2.3.2 Graduierte Exposition in der Behandlung chronischer Rückenschmerzen

Ausgehend von dem in Kapitel 2.2.1 beschriebenen Fear-Avoidance Modell und der als zentral beschriebenen Komponente der Angst haben Vlaeyen und Kollegen (Vlaeyen et al., 2001) das aus der Behandlung phobischer Ängste etablierte Therapieverfahren der graduierten Exposition in vivo auf schmerzängstliche Patienten mit chronischen Rückenschmerzen übertragen. Während einer Exposition sollen Patienten eine Bewegung durchführen, die aufgrund der dahinterliegenden Angst vor Schmerzen oder der Antizipation schädlicher Konsequenzen vermieden wird und somit über operantes Konditionieren den Kreislauf aus Angst, Vermeidung und Schmerz aufrechterhält (vgl. Kapitel 2.2.2). Die Konfrontation mit der angstbesetzten Bewegung dient dem Ziel, die Angst und das Vermeidungsverhalten zu reduzieren und dadurch langfristig ein höheres Funktionsniveau zu entwickeln. Die zuvor gelernte CS-US-Verbindung aus Bewegung (CS) und Verletzung bzw. Schmerz (US) und der häufig daraus folgenden Annahme, dass diese Bewegung schädlich für den Rücken ist, und Angst (UR) vor der Bewegung und somit Vermeidung (CR) auslöst, soll durch die korrigierende Erfahrung der Exposition (Bewegung führt nicht zu Verletzung und Angst) gehemmt werden. Lerntheoretisch wird dies über eine neue inhibitorische Assoziation von CS-noUS erklärt, die die Signalwirkung des CS reduziert und somit nicht mehr zu Angst und Vermeidungsverhalten führen soll. Neben dem Ziel der Angstreduktion sollen zudem katastrophisierende Schädlichkeitserwartungen überprüft und die neue CS-noUS-Verbindung verstärkt werden. Die Konfrontation mit der angstbesetzten Bewegung soll somit zusätzlich genutzt werden, um die Erwartungen mit dem tatsächlich eingetretenen Ergebnis zu vergleichen und dadurch neue, korrigierte Erwartungen zu entwickeln (Craske, Treanor, Conway, Zbozinek, & Vervliet, 2014; Vlaeyen, Morley, Linton, Boersma, & de Jong, 2012). Vlaeyen und Kollegen (2012) gehen davon aus, dass für ein erfolgreiches Lernergebnis sowohl die Aktivierung der Angst als auch die Überprüfung und Korrektur der Schädlichkeitsannahme wichtig sind, um die Bedrohlichkeit der angstbesetzten Bewegung zu reduzieren. Die Wirksamkeit der graduierten Exposition in vivo in der Behandlung chronischer Rückenschmerzen konnte zunächst in Einzelfallstudien und ebenfalls in randomisierten kontrollierten Studien gezeigt werden (Boersma et al., 2004; Leeuw et al., 2008; Vlaeyen, Jong, Geilen, Heuts, & van Breukelen, 2002; Vlaeyen, De Jong, Onghena, Kerckhoffs-Hanssen, & Kole-Snijders, 2002; Woods & Asmundson, 2008).

Dieses Therapieverfahren bezieht sich folglich nicht auf die Reduktion der Schmerzen, sondern auf der Reduktion der Angstreaktion und des Vermeidungsverhaltens, indem die als bedrohlich

eingeschätzte Bewegung konfrontiert wird und Schädlichkeitserwartungen mit dem tatsächlich eingetretenen Ergebnis abgeglichen und gegebenenfalls korrigiert werden. Patienten mit chronischen Rückenschmerzen sollen folglich lernen, dass sie trotz ihrer bzw. mit ihren Schmerzen Aktivitäten durchführen können und somit weniger Beeinträchtigung erleben müssen und an Lebensqualität gewinnen. Dieser Fokus, der mithilfe von Expositionen zur Verletzung der Schädlichkeitserwartungen und der Entwicklung neuer Annahmen führen soll, kann im Zwei-Prozess-Modell den akkommodativen Copingstrategien zugeschrieben werden (Rief et al., 2015).

3 DARSTELLUNG DES DISSERTATIONSVORHABENS

3.1 Relevanz und Herleitung der Fragestellungen

Aus den vorhergehenden theoretischen Ausführungen lassen sich folgende Hauptaspekte ableiten. Im Bereich der Entstehung und Aufrechterhaltung chronischer Rückenschmerzen versucht das Fear-Avoidance Modell nach Vlaeyen und Linton (2000) Chronifizierungsprozesse aus einer biopsychosozialen Perspektive zu erklären. Im Zentrum dieses Modells steht eine durch Schmerzerfahrung und katastrophisierenden Bewertungen entstandene Angst vor Schmerzen bzw. vor schmerzassoziierten Bewegungen. Aus dieser Angst resultiert wiederum Vermeidungsverhalten, das langfristig zu einem steten Rückzug aus sozialen, sportlichen und beruflichen Aktivitäten und dadurch zu negativen physiologischen sowie psychischen Folgen führt. Aus lerntheoretischer Sicht werden im FAM zunächst klassische Konditionierungsprozesse angenommen, die sich in einer CS-US-Verbindung zwischen einer mit dem Schmerz (US) assoziierten Bewegung (CS) zeigen und Angst (UR) sowie Vermeidung (CR) auslösen. Das Vermeidungsverhalten wird durch das Ausbleiben der Angst und der Schmerzen negativ verstärkt und dementsprechend über operante Konditionierungsprozesse erklärt. Trotz der angenommenen Angstkomponente im FAM konnte bislang keine Evidenz für das Vorliegen einer Angst mit phobischem Charakter, wie sie in Angststörungen zu finden ist, in Patienten mit chronischen Schmerzen gezeigt werden. Pincus und Kollegen (2014) empfehlen deshalb auch die kognitiven Komponenten des FAM zu untersuchen, um entsprechende klinische Implikationen treffen zu können und die Behandlungen und die Prävention chronischer Rückenschmerzen zu verbessern. Sowohl Patienten mit chronischen Rückenschmerzen als auch gesunde Personen berichten negative Überzeugungen gegenüber rückenbeanspruchenden Bewegungen. Diese sogenannten Fear-Avoidance Beliefs stellen eine kognitive Komponente des FAM dar und wurden in Studien als relevanter Prädiktor im Übergang von akuten zu chronischen Rückenschmerzen gefunden (Linton, Buer, Vlaeyen, & Helsing, 2000; Wertli et al., 2014). Andere Befunde geben Hinweise darauf, dass die Reduktion von FAB positive Effekte auf Behandlungsergebnisse haben kann (Brox, 2014; Woby et al., 2004). Bislang ist jedoch wenig über die Entstehungsmechanismen von FAB bekannt. Die Forschung zu Einstellungen hat evaluatives Konditionieren als einen wichtigen Lernmechanismus im Erwerb und in der Veränderung von Einstellungen gefunden. Obgleich diskutiert wird, dass evaluatives Konditionieren (EK) eine Unterform vom klassischen Konditionieren (KK) ist, gibt es Unterschiede in bestimmten Merkmalen, die für therapeutische Implikationen sowie für die Entstehung und Prävention von FAB entscheidend sein könnten. So zeigen Studien, dass inhibitorisches Lernen über CS-noUS-Verbindungen wie sie im KK angenommen werden nicht wirksam sind, sondern eine Gegenkonditionierung stattfinden muss,

um Einstellungen zu verändern. Aus diesem Grund sollte in Studie 1 experimentell untersucht werden, ob sich Einstellungen gegenüber rücken-beanspruchenden Bewegungen durch EK positiv und negativ verändern lassen. Weiterhin war von Interesse, ob sich Veränderungen gleichermaßen implizit als auch explizit zeigen.

Im Bereich der Behandlung chronischer Rückenschmerzen bietet die kognitive Verhaltenstherapie eine Vielzahl an Behandlungsmöglichkeiten an, die unterschiedliche Herangehensweisen an den Umgang mit der beeinträchtigenden Schmerzsymptomatik hat und ebenfalls anderen lern-theoretischen Annahmen unterliegt. Laut des Zwei-Prozess-Modells nach Brandtstädter und Renner (1990) können zwei gegensätzliche Copingstrategien im Umgang mit aversiven Lebensereignissen wie chronischen Erkrankungen eingesetzt werden. In Bezug auf chronische Schmerzen beschreibt assimilatives Coping jene Strategien, die zu einer Reduktion der Schmerzsymptomatik eingesetzt werden können, wohingegen akkommodatives Coping Strategien beschreibt, die den Fokus von der gewünschten Schmerzfreiheit lösen und auf neue Ziele lenken (De Vlieger et al., 2006; Schmitz et al., 1996). Einige Studien weisen darauf hin, dass assimilative Copingstrategien teilweise negative Zusammenhänge zu Depressivität, Hypervigilanz und katastrophisierendem Denken und akkommodative Copingstrategien eher einen positive Effekte zeigen (Crombez et al., 2008; L. M. McCracken, Carson, et al., 2004). Nach Brandtstädter (2007) können in komplexen Lebenssituationen beide Arten des Copings adaptiv sein und sich ergänzen. Eine noch offene Frage ist jedoch, wann eine Veränderung der Copingstrategie hilfreich ist und möglicherweise Chronifizierungsprozesse hemmen könnte (Crombez et al., 2008). Zur Untersuchung dieser Fragen bedarf es adäquater Messinstrumente. In diesem Zusammenhang erscheint der Pain Solutions Questionnaire (PaSol) ein geeigneter Fragebogen, der sowohl assimilative als auch akkommodative Copingstrategien erfasst und sowohl auf akute als auch auf chronische Schmerzen angepasst ist. Aus diesem Grund sollen Ziele der Studie 2 die Entwicklung sowie psychometrische Überprüfung einer deutschen Version des PaSol sein.

Ein von Patienten sowie Therapeuten gut akzeptiertes und für einige Schmerzstörungen (z.B. Migräne, Fibromyalgie) als wirksam belegtes Therapieverfahren aus der KVT ist Biofeedback. Mit dieser Behandlungsform werden Patienten physiologische Prozesse des autonomen oder zentralen Nervensystems über auditive, visuelle oder taktile Reize zurückgemeldet. Diese Rückmeldung soll genutzt werden, um mehr Verständnis über physiologische sowie psychologische Zusammenhänge zu entwickeln und daraufhin mehr Kontrolle darüber zu gewinnen. Biofeedback basiert auf operantem Lernen, indem mithilfe der Rückmeldung physiologische Prozesse beeinflusst werden sollen. Ziele von Biofeedback sind häufig

Entspannung über die bewusste Reduktion von Muskelanspannung und somit indirekt eine Schmerzreduktion zu erreichen. Obwohl Biofeedback bereits seit Jahrzehnten Anwendung in der Behandlung chronischer Schmerzen eingesetzt wird, ist die Wirksamkeit als Therapieverfahren bei chronischen Rückenschmerzen noch unklar. In der Literatur finden sich sowohl Befunde, welche die Wirksamkeit belegen und eine Überlegenheit gegenüber anderen KVT-basierten Behandlungen berichten, als auch Studien, in denen Biofeedback nicht hilfreich war. Da Biofeedback sowohl als eigenständige Behandlung als auch als zusätzliche Intervention einer regulären KVT-Behandlung eingesetzt wird, ist die Bestimmung der spezifischen Wirksamkeit zusätzlich erschwert. Aus diesem Grund sollte in Studie 3 der vorliegenden Dissertation die Wirksamkeit von Biofeedback als psychologische (Zusatz-)Intervention in der Behandlung chronischer Rückenschmerzen untersucht werden. Basierend auf den Ergebnissen dieser Studie werden in Studie 4 potentielle Wirkmechanismen und Limitationen dieser Behandlungsform diskutiert.

3.2 Zielsetzung und Fragestellungen des Dissertationsvorhabens

Auf Grundlage der bisherigen Forschungslage wurden im vorliegenden Dissertationsvorhaben die folgenden Zielsetzungen und Fragestellungen untersucht:

Studie 1: Können negative Einstellungen gegenüber rückenbeanspruchenden Bewegungen, im Sinne von Fear-Avoidance Beliefs, über evaluatives Konditionieren geformt werden? Wird implizit und explizit gleichermaßen gelernt?

Studie 2: Ist es hilfreich, einen akkommodativen Umgang mit chronischen Schmerzen zu lernen? Die Entwicklung einer deutschsprachigen Version des Pain Solutions Questionnaire (PaSol) zur Erfassung assimilativer und akkommodativer Copingstrategien bei Schmerzen und die Überprüfung dessen Gütekriterien.

Studie 3 & 4: Ist Biofeedback als psychologische Intervention basierend auf operantem Lernen wirksam in der Behandlung chronischer Rückenschmerzen? Wie wirkt sich eine mit Biofeedback unterstützte Behandlung auf relevante schmerzbezogene Outcomes aus? Was kann Biofeedback leisten und in welchen Bereichen stößt es an seine Grenzen?

4 ZUSAMMENFASSUNG DER STUDIEN

4.1 Studie 1: Die Rolle von evaluativem Konditionieren in der Entstehung von Fear-Avoidance Beliefs – experimentelle Studien zur Überprüfung impliziter und expliziter Einstellungen gegenüber rückenbeanspruchenden Bewegungen.

Zitation: Sielski, R., Lucke, S., Uengoer, M., Glombiewski, J.A. (submitted). Forming attitudes toward movements: The role of evaluative conditioning in the acquisition of fear-avoidance beliefs.
Manuscript submitted for publication in *PAIN*

Hintergrund. Im Fear-Avoidance Modell nach Vlaeyen und Linton (2000) wird Angst vor Bewegung als zentrale Komponente für die Entstehung und Aufrechterhaltung chronischer Rückenschmerzen angesehen. Ausgehend von einer konditionierten Angstreaktion werden klassische und operante Konditionierungsmechanismen angenommen, die dem Teufelskreis aus Angst, Schmerz und Vermeidung zugrundeliegen (den Hollander, de Jong, Volders, Goossens, Smeets, & Vlaeyen, 2010). Problematisch erscheint dieses Erklärungsmodell, da bisher keine konsistente Evidenz für eine konditionierte Angstreaktion vorliegt (Barke et al., 2012; Glombiewski et al., 2015; Pincus et al., 2010). Aufgrund dessen wird empfohlen auch kognitive Komponenten des Fear-Avoidance Modells wie Fear-Avoidance Beliefs (FAB) zu untersuchen (Woby et al., 2004). Die Relevanz von FAB in der Chronifizierung von Rückenschmerzen konnte in verschiedenen Studien gezeigt werden (Grotle et al., 2004; Wertli et al., 2014). Bislang ist wenig darüber bekannt, wie FAB entstehen und wie sie verändert werden können. Vor dem Hintergrund, dass FAB als negative Einstellungen gegenüber rückenbeanspruchenden Bewegungen beschrieben werden, erscheint ein weiterer potentieller Lernmechanismus plausibel, um die Entstehung von FAB zu erklären. In der Forschung zum Erwerb und zur Veränderung von Einstellungen gilt evaluatives Konditionieren (EK) als relevanter Lernmechanismus. Durch EK wird die Bewertung bzw. die Valenz eines Stimulus durch Paarung mit einem negativ oder positiv affektiven Stimulus verändert. Im Vergleich zu klassischem Konditionieren finden sich Unterschiede unter anderem im Hinblick auf Extinktionsprozesse (Gawronski et al., 2014; Hofmann et al., 2010). Ziel der vorliegenden experimentellen Studien war es zu überprüfen, ob sich explizite und implizite Bewertungen rückenbeanspruchender Bewegungen über EK in positive sowie negative Richtung verändern lassen. Weiterhin sollte untersucht werden, ob sich potentielle Veränderungen der Bewertung durch Gegenkonditionierung als Extinktionsprozess wieder rückgängig machen lassen. Zur Überprüfung der Hypothesen wurde zudem eine Replikationsstudie durchgeführt. Die Befunde sollen erste Hinweise darauf geben, ob EK eine Rolle in der Entstehung von FAB spielt.

Methode. Aufgrund der Ergebnisse einer Vorstudie bestehend aus einer Onlineumfrage ($N = 74$) und einem Affective Priming Paradigma ($N = 37$), in dem Bilder aus der Photograph Series of Daily Activities (PHODA; Kugler, Wijn, Geilen, de Jong, & Vlaeyen 1999) als Reizmaterial eingesetzt wurden, wurden für die Durchführung des experimentellen Paradigmas neue visuelle Stimuli entwickelt. Angelehnt an die PHODA bestehen die neu entwickelten Stimuli aus simplen, wenig detaillierten Bildern, auf denen eine typische rückenbeanspruchende Bewegung, z.B. Bücken, ausgeführt wird, die von Patienten mit chronischen Rückenschmerzen als schmerzhaft und schädlich eingeschätzt werden. Dargestellt werden ein Mann bzw. eine Frau in Form einer schwarzen Silhouette vor weißem Hintergrund. Die Auswahl der abgebildeten Bewegungen erfolgte auf Basis theoretischer und experimental-praktischer Überlegungen.

Studiendesign: Die Studie bestand aus zwei zeitlich voneinander getrennten Phasen. Zunächst bewerteten die gesunden Probanden (Experiment 1: $N = 33$; Experiment 2: $N = 50$) die Bewegungen, die auf den Stimuli dargestellt waren, auf visuellen Analogskalen (VAS) hinsichtlich der Dimensionen Valenz, Schädlichkeit sowie Schmerz. Basierend auf den Bewertungen wurden anschließend zwei als neutral bewertete Bewegungen ausgewählt und in Folge als neutrale bzw. konditionierte Stimuli (CS) verwendet. Im Labor durchliefen die Probanden zwei Lern- und zwei Testphasen. Die Lernphasen bestanden aus einem evaluativen Bild-Bild-Konditionierungsparadigma. Bilder aus dem International Affective Picture System (Lang, Bradley, & Cuthbert, 2008) dienten als unkonditionierte Stimuli (US). In der ersten Lernphase (evaluatives Konditionieren; EK Phase) wurden die verwendeten CS mit negativ oder positiv affektiven US gepaart. In der zweiten Lernphase (Gegenkonditionierung, GK Phase) wurde die CS-US-Kontingenz der ersten Lernphase umgekehrt.

Die CS-US-Zuordnung erfolgte randomisiert, sodass für jeden Probanden in der jeweiligen Lernphase eine Bewegung mit negativen US und eine Bewegung mit positiven US gepaart wurde. Auf beide Lernphasen folgte eine implizite und explizite Testphase. Ein Affective Priming Task (APT) wurde durchgeführt, um mithilfe von Reaktionszeiten als abhängige Variable implizite Veränderungen in der Bewertung der Valenz der CS zu untersuchen. Die CS wurden im APT als Primes eingesetzt. Explizite Veränderungen wurden mithilfe von VAS für die Dimensionen Valenz, Schädlichkeit und Schmerz gemessen. Dieses Studiendesign wurde in der Experiment 2 mit kleinen Abweichungen beibehalten.

Ergebnisse Experiment 1. Eine Messwiederholungs-ANOVA mit den Faktoren Kongruenz (ja/nein), Targetvalenz (positiv/negativ) und Zeitpunkt (APT-1/APT-2) ergab einen Interaktionseffekt für die drei Faktoren, $F(1,32) = 7.58$, $p < .05$. Eine genauere Prüfung mit Simple Main Effects Analysen zeigte, dass im APT-1 kürzere Reaktionszeiten in kongruenten Trials im Vergleich zu inkongruenten Trials gezeigt wurden, wenn das Target negativ war. Reaktionszeiten für Trials mit

positiven Targets unterschieden sich nicht voneinander. Nach der GK Phase wurden im APT-2 keine signifikanten Haupt- und Interaktionseffekte mehr gefunden.

Auf expliziter Ebene zeigte eine Messwiederholungs-ANOVA einen Interaktionseffekt für die Faktoren Zeitpunkt (Baseline, EK Phase, GK Phase) und die Dimension Schmerz, $F(2,59) = 5.94$, $p < .01$, und einen marginal signifikanten Interaktionseffekt für die Faktoren Zeitpunkt und die Dimension Valenz, $F(2,64) = 3.00$, $p = .057$. Beide Interaktionseffekte zeigten an, dass CS (Bewegungen), die in der EK Phase mit negativen US gepaart wurden nach der Konditionierung negativer bzw. schmerzhafter bewertet wurden im Vergleich zu CS, die mit positiven US gepaart wurden. Weder zu Baseline noch nach der GK Phasen fanden sich Unterschiede in der Bewertung der beiden CS. Des Weiteren zeigten die Ergebnisse, dass die CS im Verlauf des Experiments insgesamt negativer, schädlicher und schmerzhafter bewertet wurden und es entsprechend unabhängig von der CS-US-Kontingenz keine Veränderungen in die positive Richtung gab.

Ergebnisse Experiment 2. Eine Messwiederholungs-ANOVA mit den Faktoren Kongruenz (ja/nein), Targetvalenz (positiv/negativ) und Zeitpunkt (APT-1/APT-2) ergab nur einen Haupteffekt für den Faktor Targetvalenz ($F(1,49) = 11.44$, $p < .01$), aus dem sich ergibt, dass auf Trials mit positiven Targets schneller reagiert wurde.

Explizit zeigten sich Interaktionseffekte für die Faktoren Zeitpunkt (Baseline, EK Phase, GK Phase) und der Dimension Valenz, $F(2,64) = 6.10$, $p < .01$, sowie für die Faktoren Zeitpunkt und die Dimension Schmerz, $F(2,59) = 3.48$, $p < .05$. Ebenfalls wie in Experiment 1, basieren die Interaktionseffekte auf Unterschiede zwischen den CS nach der EK Phase. Zu Baseline und nach der GK Phase unterschieden sich die CS nicht voneinander. Zusätzliche Analysen ergaben, dass alle CS im Verlaufe des Experiments negativer, schädlicher und schmerzhafter bewertet wurden. Es wurden keine Veränderungen in positive Richtung gezeigt.

Diskussion. Die Durchführung dieser Studien hatte zum Ziel zu überprüfen, ob Einstellungen gegenüber rückenbeanspruchenden Bewegungen durch evaluatives Konditionieren experimentell in positive sowie negative Richtung verändert werden können. Weiterhin sollte untersucht werden, ob sich Veränderungen sowohl auf impliziter als auch auf expliziter Ebene zeigen. Nach den Konditionierungsphasen zeigten die Probanden in beiden Experimenten schmerzhaftere und negativere Bewertungen gegenüber rückenbeanspruchenden Bewegungen im Selbstbericht. In Experiment 1 wurde dieser Effekt ebenfalls im impliziten Maß gefunden. Eine Veränderung der Einstellungen in positive Richtung konnte in keinem der beiden Experimente weder implizit noch explizit gefunden werden. Die Ergebnisse weisen darauf hin, dass evaluatives Konditionieren ein relevanter Faktor in der Entstehung von Einstellungen zu rückenbeanspruchenden Bewegungen

sein kann und dabei neben der Valenz ebenfalls weitere Dimensionen verändern werden können. Das Fehlen einer positiven Konditionierung kann unterschiedlich erklärt werden, zum Beispiel über die Auswahl der positiven US oder einen Negativitäts-Bias (Baumeister, Bratslavsky, Finkenauer, & Vohs, 2001; Rozin & Royzman, 2001; De Sousa, Orfale, Meireles, Leite, & Natour, 2009). Die Stabilität der negativen Bewertung auch nach der Gegenkonditionierung unterstreicht zusätzlich die Hypothese, dass evaluatives Konditionieren eine Sonderform des klassischen Konditionierens darstellt und dem evaluativen Konditionieren andere Extinktionsprozesse zugrundeliegen. Zusätzlich weisen die Ergebnisse darauf hin, dass Einstellungen im Sinne der FAB ohne die Induktion von Angst oder Schmerz gebildet werden können. Aus klinischer Sicht erscheint dies besonders vor dem Hintergrund der inkonsistenten Evidenz einer phobischen Angstkomponente in Patienten mit chronischen Rückenschmerzen nennenswert und hebt die Relevanz von kognitiven Faktoren hervor. Ferner geben die Ergebnisse Hinweise darauf, dass Einstellungen gegenüber rückenbeanspruchenden Bewegungen leicht gelernt, aber schwierig verändert werden können, sodass Interventionen in der klinischen Praxis inhibitorisches Lernen verstärken sollten, um (langfristig) wirksame Therapieerfolge zu erreichen (Craske et al., 2008, 2014).

4.2 Studie 2: Entwicklung und psychometrische Überprüfung einer deutschen Version des Pain Solutions Questionnaire (PaSol) zur Erfassung assimilativer und akkommodativer Copingstrategien bei chronischen Schmerzen.

Zitation: Sielski, R., Glombiewski, J.A., Rief, W., Crombez, G., Barke, A. (submitted). Cross-cultural adaptation of the German Pain Solutions Questionnaire: an instrument to measure assimilative and accommodative coping in response to chronic pain. Manuscript submitted for publication in *Journal of Pain Research*

Hintergrund. Der Umgang mit chronischen Schmerzen ist schwierig, sodass viele betroffene Patienten häufig hartnäckig nach einer Lösung suchen, um wieder schmerzfrei zu sein. Dieses Ziel wird unter hoher Anstrengung und mit viel Aufwand verfolgt, führt jedoch nur selten zum Erfolg und ist im Fall wiederholter Misserfolge mit geringerer Schmerztoleranz, mehr Sorgen und Angst assoziiert. In dem von Brandtstädter und Renner (1990) entwickelten Zwei-Prozess-Modell werden zwei Modi zum Umgang mit aversiven Lebensereignisse wie chronischen Schmerzen beschrieben. Assimilatives Coping bezieht sich dabei auf Copingstrategien, die abzielen das aversive Ereignis – in diesem Fall die Schmerzen – zu reduzieren. Im Gegensatz dazu steht akkomodatives Coping, das eine Umlenkung der eigenen Ziele und Werte beschreibt und somit einen flexibleren Umgang mit den gegebenen Lebensumständen bedeutet. In Bezug auf chronische Schmerzen konnten Studien zeigen, dass akkommodative Strategien wie die Akzeptanz der Schmerzen sowie das Erarbeiten neuer Ziele, positive Auswirkungen haben können (McCracken, Vowles, & Eccleston, 2004). Diese Befunde geben Hinweis darauf, dass ein Wechsel von assimilativen zu akkomodativen Copingstrategien bei chronischen Rückenschmerzen sinnvoll sein kann. Auf Basis des Zwei-Prozess-Modells wurde der Pain Solutions Questionnaire (PaSol; De Vlieger et al., 2006) entwickelt. Der PaSol erfasst beide Copingstrategien und ist sowohl für akute als auch chronische Schmerzen angepasst. Weiterhin sollte der Fragebogen relativ unabhängig zu schmerzrelevanten Konstrukten wie katastrophisierendem Denken sein. Bislang gibt es noch keine validierte deutsche Version des PaSol. Ziel der vorliegenden Studie ist die Entwicklung einer deutschsprachigen Version des PaSol sowie der Überprüfung seiner psychometrischen Qualitäten. Zusätzlich soll die Sensitivität der Subskalen auf Therapieerfolg nach einer kognitiv-verhaltenstherapeutischen Intervention untersucht werden.

Methode. Der PaSol besteht aus 14 Items, die auf einer sieben-stufigen Antwortskala (0 = Trifft überhaupt nicht zu; 6 = Trifft vollkommen zu) verschiedene Einstellungen zu Copingstrategien bei Schmerzen erfassen. Die Originalautoren berichten vier Subskalen, von denen *Problem Solving*

sowie *Belief in a Solution* zu assimilativem Coping und *Meaningfulness of Life Despite of Pain* sowie *Acceptance of the Insolubility of Pain* zu akkomodativem Coping gezählt werden (De Vlieger et al., 2006). Die Auswertung erfolgt auf Subskalenebene und wird über die Summe der Items pro Subskala berechnet. Die Entwicklung einer deutschsprachigen Version orientierte sich an internationalen Richtlinien zur transkulturellen Adaption von Selbstbeurteilungsinstrumenten (Beaton, Bombardier, Guillemin, & Ferraz, 2000). Die psychometrischen Eigenschaften der deutschsprachigen Version wurden an einer Stichprobe von 165 Patienten mit chronischen Rückenschmerzen, die sich in ambulanter oder stationärer Behandlung befanden, überprüft. Daten von 95 Patienten, die eine ambulante kognitiv-verhaltenstherapeutische Behandlung gemacht haben, wurden zur Berechnung der Sensitivität des Fragebogens auf Therapieerfolg analysiert. Die Überprüfung der psychometrischen Eigenschaften erfolgte über Itemanalysen, u.a. Itemschwierigkeit, und Untersuchungen der internen Konsistenz mit Cronbachs α . Zur Überprüfung der Validität wurden schmerzrelevante Fragebögen wie der Pain Disability Index (PDI) oder die Hospital Anxiety and Depression Scale (HADS) eingesetzt. Die Überprüfung der Faktorstruktur erfolgte über eine explorative Faktoranalyse. Zusätzlich wurde untersucht, ob die Subskalen des Fragebogens inkrementelle Varianz über demographische sowie Schmerzcharakteristika auf Beeinträchtigung (PDI) oder Depressivität (HADS) aufklärt. Die Sensitivität des Fragebogens auf Therapieerfolg wurde mit hierarchischen multiplen Regressionen berechnet. Therapieerfolg wurde über eine Verbesserung von mindestens 30% im PDI definiert.

Ergebnisse. Die deutschsprachige Version des PaSol zeigte gute psychometrische Eigenschaften. Sowohl die mittleren Itemschwierigkeiten ($p_i = .62-.79$) als auch die internen Konsistenzen für die Subskalen (Cronbachs $\alpha = .71-.84$) sind akzeptabel bis gut. Die explorative Faktoranalyse ergab die von den Originalautoren vorgeschlagene Vier-Faktor-Lösung mit derselben Item-Skala-Zuordnung für jeden der Faktoren. Eigenwerte lagen zwischen 2.50 und 1.45 und erklärten 56.30% der Gesamtvarianz. Die mittlere Itemkommunalität lag bei 0.56. Korrelationen zu schmerzrelevanten Messinstrumenten waren im kleinen bis mittleren Bereich. Die Subskalen *Meaningfulness of Life Despite Pain* und *Solving Pain* klärten nach Kontrolle für demographische und Schmerzcharakteristika zusätzliche Varianz auf Depressivität (HADS) auf. Hinsichtlich schmerzbedingter Beeinträchtigung (PDI) klärte die Subskala *Meaningfulness of Life Despite Pain* zusätzliche Varianz auf, wenn Schmerzdauer nicht mit in die Analysen aufgenommen wurde. Für die Subskala *Meaningfulness of Life Despite Pain* konnte eine signifikante Interaktion mit dem Faktor Zeitpunkt (vor Therapie/nach Therapie) hinsichtlich Therapieerfolg gezeigt werden, $F(1,64) = 5.49, p = .022$.

Diskussion. Es ist gelungen eine deutschsprachige Version des Pain Solutions Questionnaires mit guten psychometrischen Eigenschaften zu entwickeln. Die Vier-Faktor-Struktur und somit die

vorgeschlagenen Subskalen konnten repliziert werden und weist ähnlich gute psychometrische Qualitäten zu Ergebnissen aus anderen Studien auf (De Vlieger et al., 2006; Crombez et al., 2008). Die klein bis mittleren Korrelationen zu schmerzrelevanten Konstrukten bestätigen die relative Unabhängigkeit des PaSol, die in der Entwicklung des Fragebogens angenommen wurde. Die Subskalen *Solving Pain* und *Acceptance of the Insolubility of Pain* sagten Depressivität vorher. Höhere Werte auf der Subskala *Solving Pain* waren demnach mit mehr Depressivität assoziiert. Für die Subskala *Acceptance of the Insolubility of Pain* waren hohe Werte mit weniger Depressivität verbunden. Dieses Ergebnis stimmt insofern mit der Literatur zu assimilativen und akkommodativen Copingstrategien bei chronischem Schmerz überein, als dass die beständige Suche nach einer Lösung für die Schmerzen mit vermehrten negativen Symptomen assoziiert ist wohingegen für die Akzeptanz von Schmerzen positive Effekte gefunden wurden. Die Befunde zur Sensitivität des Fragebogens geben zudem erste Hinweise, dass die Subskala *Meaningfulness of Life Despite Pain* sensitiv ist, um Veränderungen während einer Therapie zu messen und ein positiver Zusammenhang zwischen Therapieerfolg und dieser Subskala besteht. Weitere Studien mit größeren Stichproben sollten die Ergebnisse der Faktorstruktur mit einer konfirmatorischen Faktoranalyse überprüfen und vergleichen. Ein spezifischer akzeptanzbasierter Fragebogen wie der Chronic Pain Acceptance Questionnaire (CPAQ; McCracken et al., 2004) sollte zusätzlich eingesetzt werden, um die Validität des PaSol genauer zu untersuchen. In Bezug auf den Anwendungsbereich liegt mit dem PaSol nun ein geeignetes Instrument vor, mit dem sowohl assimilative als auch akkommodative Copingstrategien untersucht werden können.

4.3 Studie 3: Die Wirksamkeit von Biofeedback in der Behandlung chronischer Rückenschmerzen

Zitation: Sielski, R., Rief, W., Glombiewski, J.A. (2016). Efficacy of Biofeedback in Chronic back Pain: a Meta-Analysis. *Int. J. Behav. Med.* doi:10.1007/s12529-016-9572-9

Hintergrund. Chronische Rückenschmerzen gehören mit einer Lebenszeitprävalenz von 30-40% zu den größten Herausforderungen im Gesundheitswesen westlicher Gesellschaften und führen zu negativen Konsequenzen für die betroffenen Personen und hohen sozioökonomischen Kosten (Breivik et al., 2006; Maniadakis & Gray, 2000). Personen, die von chronischen Rückenschmerzen betroffen sind, berichten über bedeutende Beeinträchtigungen in ihrer Alltagsaktivität, ihrem sozialen Funktionsniveau sowie ihrer Arbeits- und Erwerbstätigkeit, die sich in einer generell niedrigeren Lebensqualität ausdrücken und neben der Schmerzsymptomatik häufig mit Depression, niedrigen Selbstwirksamkeitserwartungen sowie erhöhter muskulärer Anspannung einhergehen (Scholich, Hallner, Wittenberg, Hasenbring, & Rusu, 2012). Effektive und ökonomische Behandlungen chronischer Rückenschmerzen sowie der damit einhergehenden Belastungen sind nötig. Biofeedback stellt dabei ein Behandlungselement der kognitiven Verhaltenstherapie dar, das sich physiologischen und psychologischen Methoden bedient und sowohl als eigenständige Intervention als auch als Teilelement einer Behandlung eingesetzt werden kann. Während Biofeedbacksitzungen werden Patienten auditive, visuelle oder taktile Reize als Rückmeldung über physiologische Prozesse ihres autonomen oder zentralen Nervensystems, z.B. Muskelanspannung oder elektrodermale Aktivität, dargeboten, um diese Prozesse besser wahrnehmen und kontrollieren zu können. Die Einflussnahme auf physiologische Prozesse durch Rückmeldung wird über operantes Lernen erklärt (Schwartz & Schwartz, 2003). Studien zur Wirksamkeit von Biofeedback in der Behandlung chronischer Rückenschmerzen bislang inkonsistente Befunde und konnten kein abschließendes Bild ergeben.

Methode. Um die Wirksamkeit von Biofeedback in der Behandlung chronischer Rückenschmerzen auf die von der „Initiative on Methods, Measurement and Pain Assessment in Clinical Trials“ (IMMPACT) empfohlenen Zielgrößen Schmerzintensität, Depression, Beeinträchtigung, Selbstwirksamkeitserwartungen sowie muskuläre Anspannung miteinander zu vergleichen, wurden in einer umfangreichen Suche in PubMed, PsycINFO und Cochrane Library insgesamt 412 relevante Studien identifiziert. Von diesen Studien konnten nach Überprüfung der Ein- und Ausschlusskriterien 21 Studien eingeschlossen werden. Für den Einschluss der Studien wurde unter anderem ein Mindestanteil von 25% Biofeedback an der Gesamtbehandlungszeit und ausreichend Daten zu mindestens einer der oben genannten Zielgrößen, um Effektstärken berichten zu können,

vorausgesetzt. Effektstärken (Hedges' g) wurden für prä-post sowie für prä-follow-up gerechnet und metaanalytisch mithilfe eines Random-Effects-Modells integriert. Für Studien mit Kontrollgruppen (Aktive KG, Warteliste) wurden weiterhin kontrollierte Effektstärken berechnet. Zusätzlich wurde der Einfluss potentieller Moderatoren, z.B. Qualität der Studie oder Stichprobengröße, auf die verschiedenen Zielgrößen untersucht.

Ergebnisse. Die Effektstärken für die Wirksamkeit von Biofeedback für prä-post Daten waren klein bis mittel, jedoch für alle Zielgrößen signifikant und robust (Schmerzintensität: $g = 0.60$; Depression: $g = 0.40$; Beeinträchtigung: $g = 0.49$; Selbstwirksamkeit: $g = 0.41$; Reduktion muskulärer Anspannung: $g = 0.44$). Diese Ergebnisse blieben nach einem Follow-Up von durchschnittlich 8 Monaten stabil. Bis auf die Zielgröße Beeinträchtigung konnten auch für Studien mit Kontrollgruppen kleine bis mittlere kontrollierte Effektstärken gezeigt werden. Die Ergebnisse gaben weiterhin Hinweise darauf, dass Behandlungen mit Biofeedback besonders wirksam in der Reduktion muskulärer Anspannung sind verglichen mit ähnlichen Behandlungen ohne Biofeedback oder Wartekontrollgruppen. Moderator-Analysen stellten Studienqualität, Stichprobengröße, Anteil von Biofeedback an der Gesamtbehandlungszeit sowie Behandlungsdosis von Biofeedback (in Minuten) als Moderatoren für verschiedene Zielgrößen heraus. Abschließende Subanalysen deuteten an, dass Behandlungen mit Biofeedback im Vergleich zu Behandlungen ohne Biofeedback häufiger zu einer klinisch signifikanten Schmerzreduktion (30%-Kriterium) führten.

Diskussion. Die Ergebnisse, dass Behandlungen mit Biofeedback – sowohl als Einzel- als auch als Teilintervention einer Gesamtbehandlung – auf chronische Rückenschmerzen kleine bis mittlere Effekte haben, stehen im Einklang mit der bestehenden Literatur zu psychologischen Interventionen bei chronischen Rückenschmerzen (Henschke et al., 2011; Hoffman et al., 2007). Im Gegensatz zu der vorliegenden Meta-Analyse wurden in bisherigen Reviews jedoch respondente Verfahren zusammengefasst, sodass die spezifische Wirksamkeit von Biofeedback gegenüber beispielsweise Entspannungsverfahren nicht untersucht wurde. Die Ergebnisse für die kontrollierten Effektstärken zeigten jedoch die Relevanz für eine Unterscheidung innerhalb responder Verfahren an. Die vorliegenden Ergebnisse weisen auf eine kurz- und langfristige Überlegenheit von Behandlungen mit Biofeedback gegenüber anderen responder Verfahren wie Entspannung in der Behandlung chronischer Rückenschmerzen hin. Nichtsdestotrotz zeigte sich, dass die Forschung zu Biofeedback bei chronischen Rückenschmerzen insgesamt spärlich ist, sodass die vorgefundenen Effekte, insbesondere zur Langzeitwirksamkeit, mit Bedacht interpretiert werden sollten. Die Effekte wurden auf statistische Signifikanz geprüft, können jedoch nicht äquivalent auf klinisch signifikante Verbesserung übertragen werden. Weiterhin

wurden nur veröffentlichte Studien und methodisch heterogene und somit auch qualitativ schwächere Studien mit in die Analysen aufgenommen, um genügend Daten zur Berechnung zu haben. Da Studienqualität als signifikanter Moderator identifiziert wurde, muss dies als Limitation dieser Meta-Analyse betrachtet werden. Die unterschiedlichen Definitionen, Verfahren, Modalitäten sowie Behandlungsdosen führen weiterhin dazu, dass die spezifische Wirksamkeit von Biofeedback nur im Ansatz geklärt werden konnte. Aus wissenschaftlicher Sicht fehlen insbesondere randomisiert-kontrollierte Studien sowie Daten auf Verhaltensmaße wie zum Beispiel Krankheitsverhalten. Weiterhin sollte untersucht werden, welche Wirkweisen in der Biofeedbackbehandlung eine zentrale Rolle spielen. Aufgrund der potentiellen langfristigen Wirksamkeit von Biofeedback auf verschiedene schmerzrelevante Zielgrößen kann jedoch empfohlen werden, in der klinischen Praxis Biofeedback als psychologisches Verfahren einzusetzen. Im Vergleich zu langfristiger Einnahme von Opioiden kann von einer höheren Effektivität sowie geringeren Nebenwirkungen ausgegangen werden kann.

4.4 Studie 4: Biofeedback als psychologisches Behandlungsverfahren für chronische Rückenschmerzen.

Zitation: Sielski, R., Glombiewski, J.A. (2016). Biofeedback as a psychological treatment option for chronic back pain. *Pain Management*. doi:10.2217/pmt-2016-0040

Hintergrund. Biofeedback wird bereits seit Jahrzehnten als psychologisches Verfahren in der Behandlung chronischer Rückenschmerzen eingesetzt und stetig weiterentwickelt. Dabei ist das Prinzip von Biofeedback gleich geblieben: die Rückmeldung physiologischer Prozesse des autonomen oder zentralen Nervensystems, die oft nicht bewusst wahrgenommen werden (Neblett, 2016). Mithilfe von Biofeedback sollen Patienten über operantes Lernen, physiologische Prozesse besser wahrnehmen und selbst regulieren können. In der Behandlung chronischer Rückenschmerzen wird Biofeedback dabei in verschiedenen Modalitäten sowohl als eigenständige Behandlung als auch als zusätzliche Intervention zu einer Standardbehandlung eingesetzt. Obwohl Biofeedback nicht als neues psychologisches Behandlungsverfahren für chronische Rückenschmerzen gilt, sind noch einige Fragen ungeklärt, die in der vorliegenden Studie diskutiert werden.

1. Ist Biofeedback eine sinnvolle und wirksame Intervention in der Behandlung chronischer Rückenschmerzen?

Die Erfassung der spezifischen Wirksamkeit von Biofeedback in der Behandlung chronischer Rückenschmerzen ist schwierig, weil Biofeedback in den meisten Fällen als eine von vielen Interventionen einer kognitiven Verhaltenstherapie eingesetzt oder in Reviews mit anderen verhaltenstherapeutischen, respondenten Verfahren wie Entspannung zusammengefasst wird. In einer kürzlich erschienenen Meta-Analyse (Sielski, Rief, & Glombiewski, 2016; s. Studie 3) wurde dieser Schwierigkeit entgegengewirkt, indem nur Studien mit einem Mindestanteil von 25% Biofeedback an der Gesamtbehandlungszeit in die Analysen eingeschlossen wurden. Die Effektstärken für verschiedene schmerzrelevante Zielgrößen lagen im kleinen bis mittleren Bereich. Diese Ergebnisse waren sowohl für kurz- als auch langfristige Wirksamkeit vergleichbar robust. Die Befunde geben Hinweise darauf, dass Biofeedback als eigenständige oder zusätzliche Intervention einer Behandlung wirksam und teilweise anderen Behandlungsformen, z.B. Entspannung oder Psychoedukation, überlegen sein kann. Forschung mit größeren Stichproben und randomisiert-kontrollierte Studien sollte jedoch weiter durchgeführt werden, um diese Ergebnisse zu sichern.

2. Was sind die Wirkmechanismen von Biofeedback?

Forschung zu Kopfschmerzen und Migräne konnte Selbstwirksamkeit als zentralen Wirkmechanismus von Biofeedback herausstellen (Nestoriuc & Martin, 2007; Nestoriuc et al., 2008). Für chronische

Rückenschmerzen ist die Forschungslage bislang wenig eindeutig. In der bereits genannten Meta-Analyse konnten die größten Effekte auf die Zielgröße Reduktion muskulärer Anspannung gefunden werden. Besonders vor dem Hintergrund, dass Patienten mit chronischen Rückenschmerzen stärkere psychophysiologische Reaktionen zeigen (Glombiewski et al., 2015), erscheint die Reduktion muskulärer Anspannung ein plausibler Wirkmechanismus für dieses Störungsbild zu sein. Entspannungsverfahren oder physiotherapeutische Maßnahmen setzen jedoch ebenfalls an diesen Mechanismen an und waren Behandlungen mit Biofeedback in der vorgestellten Meta-Analyse unterlegen. Zusätzlich konnte eine Studie von Kapitza und Kollegen (2010) zeigen, dass der Einsatz von Atembiofeedback als Placebo positive Effekte auf schmerzrelevante Faktoren hatte. Diese Ergebnisse weisen darauf hin, dass sich die Wirksamkeit von Biofeedback nicht ausreichend durch die alleinige Reduktion von muskulärer Anspannung erklären lassen kann.

3. Gibt es Einschränkungen in der Behandlung von chronischen Rückenschmerzen mit Biofeedback?

Studien konnten für verschiedene schmerzrelevante Zielgrößen die Wirksamkeit von Biofeedback als psychologisches Behandlungsverfahren zeigen. Eine in der Psychotherapie und für Patienten mit chronischen Rückenschmerzen wichtige Belastung stellt schmerzbedingte Beeinträchtigung dar. Für diese Zielgröße scheint die Wirksamkeit von Biofeedback eingeschränkt zu sein und sollte durch Expositionsverfahren oder graduierten Aktivitätenaufbau ergänzt werden (Boersma et al., 2004; Woods & Asmundson, 2008). Weitere Schwierigkeiten sind in der ambulanten Versorgung bzw. Inanspruchnahme von Biofeedback zu finden. Trotz hoher Akzeptanz von Patienten und kurz- und langfristiger Wirksamkeitshinweise wird dieses psychologische Verfahren aufgrund technischer und finanzieller Herausforderungen auf Therapeutenseite sowie der Vielzahl an Einsatzmöglichkeiten und Modalitäten nur selten in der Praxis eingesetzt (Rief & Birbaumer, 2011; Schwartz & Schwartz, 2003).

5 ZUSAMMENFASSENDE DISKUSSION UND AUSBLICK

In der vorliegenden Dissertation ist es gelungen, ein experimentelles Paradigma zu entwickeln, in dem erstmals evaluative Konditionierungsprozesse zur Veränderung von impliziten und expliziten Einstellungen gegenüber rückenbeanspruchenden Bewegungen im Sinne der Fear-Avoidance Beliefs eingesetzt und überprüft wurden. Zusätzlich wurde neues Stimulusmaterial entwickelt, um die Fragestellungen der Studie 1 adäquat überprüfen zu können. Die Auswahl und Entwicklung der eingesetzten Stimuli orientierte sich dabei an theoretischen sowie experimentell-praktischen Merkmalen, die im Bereich der Forschung zu chronischen Rückenschmerzen als relevant betrachtet werden. Um die Interpretierbarkeit der Ergebnisse zu verbessern, wurde das experimentelle Paradigma in einer Replikationsstudie wiederholt an einer gesunden Stichprobe durchgeführt und wie es für psychologischen Fragestellungen empfohlen wird (Asendorpf et al., 2012). Die Ergebnisse geben Hinweise darauf, dass die Einstellungen von rückenbeanspruchenden Bewegungen durch evaluatives Konditionieren in negative Richtung verändert werden können. Die Probanden bewerteten die dargestellten Bewegungen im Selbstbericht (explizit) nach der Akquisition negativer und schmerzhafter. Implizit scheint die Veränderung der Einstellungen über evaluatives Konditionieren komplexer zu sein und konnte nur im ersten Experiment im impliziten Maß, dem Affective Priming Task, in negative Richtung gefunden werden. Konsistent über beide Experimente zeigten sich keine Veränderungen der Einstellungen in positive Richtung. Die Ergebnisse beider Experimente lassen darauf schließen, dass Fear-Avoidance Beliefs über evaluatives Konditionieren auch ohne die Induktion von Schmerz oder Angst entstehen können. Die Befunde liefern einen weiteren Erklärungsansatz, warum Fear-Avoidance Beliefs auch in gesunden Populationen berichtet werden. Die nur in negative Richtung gefundenen Einstellungsveränderungen lassen zudem auf einen Negativitätsbias schließen. Dieser deutet darauf hin, dass negative, bedrohliche Einstellungen gegenüber rückenbeanspruchenden Bewegungen schneller und robuster gelernt werden als positive, protektive Einstellungen.

Die Art des Umgangs mit Schmerzen wird als relevantes Merkmal im Übergang von akuten zu chronischen Schmerzen und ebenfalls in der Schwere der Symptomatik bei einer bereits vorhandenen Chronifizierung diskutiert (Crombez, Eccleston, Van Hamme, et al., 2008; McCracken, Carson, et al., 2004; Van Damme, Crombez, & Eccleston, 2008). Der Pain Solutions Questionnaire (PaSol; De Vlieger et al. 2006) ist ein Fragebogen, das assimilative sowie akkommodative Copingstrategien bei akuten und chronischen Schmerzen erfasst und erscheint als adäquates Messinstrument, um die diskutierten Fragestellungen untersuchen zu können. In Studie 2 wurde erfolgreich eine deutsche Version des PaSol entwickelt, deren psychometrische Überprüfung auf ein reliables und valides Instrument schließen lässt, um assimilative und akkommodative

Copingstrategien im Umgang mit chronischen Schmerzen zu erfassen. Die von den Originalautoren des PaSol vorgeschlagene Vier-Faktor-Struktur wurde in den Daten wiedergefunden und beibehalten. Zusätzlich konnte gezeigt werden, dass insbesondere die zum akkommodativen Coping zugehörigen Subskalen sensitiv für Veränderungen durch Psychotherapie sind.

Patienten mit chronischen Rückenschmerzen sollen mithilfe von kognitiver Verhaltenstherapie (KVT) einen verbesserten Umgang mit ihrer Symptomatik lernen. Dabei werden in der KVT verschiedene Ansätze angeboten, mithilfe derer assimilative oder akkommodative Copingstrategien gelernt werden sollen und denen andere Lernmechanismen unterliegen. Ein bereits für Kopfschmerzen und Fibromyalgie als wirksam belegtes Therapieverfahren, das auf operantem Lernen basiert, ist Biofeedback (Glombiewski et al., 2013; Nestoriuc et al., 2008). In Studie 3 konnte für Biofeedback als eigenständige Therapie oder zusätzliche Intervention in der Behandlung chronischer Rückenschmerzen eine meta-analytische Übersicht über die Wirksamkeit bezüglich Symptomverbesserungen auf schmerzrelevante Zielvariablen gegeben werden. Hierbei zeigten sich für alle Zielvariablen kleine bis mittlere Effekte. Diese Effekte blieben auch nach einem Follow-Up von durchschnittlich acht Monaten und mit Ausnahme für die Zielvariable Beeinträchtigung auch für kontrollierte Effektstärken relativ stabil. Behandlungen mit Biofeedbackanteil zeigten sich insbesondere auf die Reduktion von muskulärer Anspannung wirksamer im Vergleich zu Behandlungen ohne Biofeedback. Die Ergebnisse lassen den Schluss zu, dass (zusätzlich zur Standardbehandlung durchgeführtes) Biofeedback als operantes Verfahren wirksam verschiedene Symptome von Patienten mit chronischen Rückenschmerzen reduzieren kann.

Ein Überblick über Biofeedback in der Behandlung chronischer Rückenschmerzen konnte in Studie 4 gegeben werden. Biofeedback bietet demnach eine Vielfalt an Modalitäten und Feedbackmöglichkeiten und kann verschiedene physiologische Prozessen rückmelden, die sowohl für die Diagnostik als auch als Intervention genutzt werden können. Der angenommene Wirkmechanismus über Entspannung der Muskulatur positiv auf andere schmerzbezogene Symptome zu wirken, scheint nach neueren Studien nicht mehr haltbar zu sein. Die Wirksamkeit von Biofeedback konnte belegt werden. Trotzdem sollten insbesondere stark beeinträchtigte Patienten zusätzliche Interventionen angeboten bekommen, um der heterogenen Schmerzsymptomatik gerecht zu werden.

5.1 Einschränkungen

Bei der Interpretation der Ergebnisse der präsentierten Studien sind Einschränkungen zu berücksichtigen. In Studie 1 ist zunächst die Auswahl des Stimulusmaterials zu beachten. Aus theoretischer sowie praktischer Sicht erscheinen die entwickelten neutralen Stimuli adäquat, jedoch

ist trotzdem möglich, dass die Bewertungen der Stimuli nicht auf die dargestellten Bewegungen bezogen wurden und somit nicht Einstellungen gegenüber rückenbeanspruchenden Bewegungen gemessen wurden. Weiterhin ist die Interpretation der inkonsistenten Ergebnisse im impliziten Maß schwierig, da es für diese abhängige Variable keine Daten zu Baseline gab. Aufgrund dessen kann nicht sichergestellt werden, ob die ausgewählten Stimuli zu Baseline tatsächlich neutral waren. Die Selbstauskünfte auf expliziter Ebene lassen zwar darauf schließen, können jedoch von der impliziten Bewertung abweichen (Gheldof et al., 2004). In einer weiteren Durchführung sollte bestenfalls schon vor der Akquisitionsphase ein impliziter Test durchgeführt werden, um mögliche Veränderungen nach der Akquisition feststellen zu können. Weiterhin muss die Auswahl der US kritisch betrachtet werden. In beiden Durchführungen des experimentellen Paradigmas wurden US nach Normen aus dem Originalmanual des IAPS (Lang, Bradley, & Cuthbert, 2008) als Grundlage herangezogen. Im zweiten Experiment wurde nachträglich die Valenz und das Arousal der US erfasst und keine signifikanten Unterschiede für beide Dimensionen gefunden werden. Nichtsdestotrotz sollten Folgestudien für die Stichprobe angepasste und durch die Probanden ausgewählte US verwenden, um sicherzustellen, dass diese sowohl als positive als auch negative Stimuli wahrgenommen werden. Das ausgelöste Arousal könnte zusätzlich psychophysiologisch über den Hautleitwert erfasst werden.

Die Entwicklung der deutschen Version des Pain Solutions Questionnaires und der Überprüfung der psychometrischen Qualitäten (Studie 2) enthält ebenfalls Limitationen. Die gefundene Vier-Faktor-Struktur wurde nur explorativ analysiert und konnte aufgrund der geringen Stichprobengröße nicht konfirmatorisch bestätigt werden. Weitere Studien sollten die gefundene Faktorstruktur genauer untersuchen und die Güte des Modells überprüfen. Zusätzlich liegen keine Daten zur Retestreliabilität vor. Eine weitere Einschränkung ist die Auswahl der für die Analyse der Validität eingesetzten Fragebögen. In diesem Bereich fehlt der Studie ein Fragebogen, der akzeptanzbasierte Strategien abfragt. Der Chronic Pain Acceptance Questionnaire (McCracken, Vowles, & Eccleston, 2004) wäre hier ein geeigneter Fragebogen, um zu untersuchen, ob die den akkommodativen Copingstrategien zugeordneten Subskalen tatsächlich dieses Konstrukt erfassen. Weiterhin basieren die Ergebnisse lediglich auf Selbstbeurteilungsfragebögen. Insbesondere da der PaSol Copingstrategien misst, wäre eine Erfassung des tatsächlichen Copingverhaltens im Alltag oder zunächst unter Laborbedingungen, z.B. mit dem Behavioral Avoidance Test – Back Pain for CLBP patients (Holzapfel, Riecke, Rief, Schneider, & Glombiewski, 2016) wünschenswert. Da die Stichprobe nur aus Patienten mit chronischen Rückenschmerzen bestand, können zudem keine Rückschlüsse auf Populationen mit akuten Rückenschmerzen gezogen werden, sodass unklar ist, ob der Fragebogen auch für diese Stichprobe reliabel und valide ist.

Die Ergebnisse der Meta-Analyse (Studie 3) sind mit den für meta-analytische Untersuchungen üblichen Einschränkungen zu interpretieren (Hofmann & Smits, 2008). Die Grundlage der Analysen sowie der Diskussion bilden die eingeschlossenen Studien, die anhand der apriori definierten Ein- und Ausschlusskriterien zunächst subjektiv sind und zu andere Interpretationen führen können (Studie 3 & Studie 4). Die Aufnahme von nur publizierten Studien kann trotz der Ergebnisse des Fail-safe N , die keinen Publikationsbias ergeben haben, einen potentiellen Einfluss von Befunden aus nicht publizierten Studien nicht ausschließen. Die eingeschlossenen Studien variierten in ihrer Qualität und wurden aufgrund der geringen Anzahl an Studien zu Biofeedback bei chronischen Rückenschmerzen auch bei niedriger Qualität in die Analysen eingeschlossen. Die Qualität der Studien wurde jedoch als Moderator anhand modifizierter Jadad Kriterien erfasst und berechnet (Jadad et al., 1996). Die Heterogenität der Studien zeigte sich weiterhin in der Definition von Biofeedback, der beschriebenen Diagnosen, den zusätzlichen Behandlungselementen sowie dem Anteil von Biofeedback an der Gesamtbehandlungszeit. Aufgrund dessen kann lediglich eine generelle Wirksamkeit von Biofeedback in der Behandlung chronischer Rückenschmerzen interpretiert werden. Unklar bleibt weiterhin ob ein bestimmtes Biofeedbackverfahren wirksamer bei einer spezifischen chronischen Rückenschmerzsymptomatik ist. In Subanalysen konnten Hinweise gefunden werden, dass die statistisch signifikanten Verbesserungen in Behandlungen mit Biofeedback auch häufiger zu klinisch signifikanter Verbesserung der Symptomatik führen, jedoch sollten dieses Ergebnisse als vorläufig und mit Vorsicht interpretiert werden. Gleiches gilt für die Ergebnisse zu Follow-Up. Für die Berechnung der Effektstärken zu Follow-Up sank die Anzahl eingeschlossener Studien und ist vor allem auf die Zielgröße Reduktion muskulärer Anspannung mit $k = 3$ Studien sehr klein.

5.2 Perspektiven für die Forschung und Implikationen für die klinische Praxis

Aus den Ergebnissen der vorliegenden Dissertation lassen sich mehrere Ansatzpunkte für weitere Forschung ableiten. Die Befunde aus Studie 1 zeigen, dass evaluative Konditionierungsprozesse in der Entstehung von Fear-Avoidance Beliefs relevant sein können, sodass auch evaluatives Konditionieren weiter im Bereich der Schmerzforschung untersucht werden sollte. Die Befunde beider Experimente werfen die Frage auf, warum keine Veränderungen in positiver Richtung stattgefunden haben und ob rückenbeanspruchenden Bewegungen überhaupt positive bzw. protektive Eigenschaften attribuiert werden können. Aus diesem Grund sollte in Folgestudien besonders die Auswahl der positiven US berücksichtigt werden und auch die Anzahl der Trials in der Akquisitionsphase beachtet werden. Um einem Negativitätsbias entgegenzuwirken, schlagen Baumeister und Kollegen (2001) beispielsweise vor, für die Akquisition positiver CS-US-Verbindungen mehr Trials darzubieten. Dies wäre zudem wichtig, um eine erfolgreiche Gegenkonditionierung zu erreichen; andernfalls sollten auch weitere

Methoden überprüft werden, um inhibitorisches Lernen zu ermöglichen. Die Unterschiede im impliziten und expliziten Maß sollten dazu anregen, in weiteren Studien beide Ebenen zu erheben, um ein vollständigeres Bild über Fear-Avoidance Beliefs zu bekommen. Hier könnten auch weitere implizite Maße, wie z.B. der Implicit Association Task (Greenwald, McGhee, & Schwartz, 1998), eingesetzt werden. Zusätzlich lassen die Ergebnisse den Schluss zu, dass Replikationsstudien besonders wichtig für neu entwickelte Paradigmen oder eingesetzte Methoden sind, damit keine voreiligen Schlüsse gezogen werden oder um mehr Hinweise auf Moderatoren bzw. Mediatoren zu bekommen. Stichprobencharakteristika wie Vorerfahrungen mit Rückenschmerzen oder Depressivität sollten in folgenden Experimenten genauer untersucht werden, um den möglichen Einfluss dieser Merkmale zu untersuchen und mögliche Risikofaktoren ausmachen zu können. Von Interesse wären ebenfalls Untersuchungen an Stichproben mit Patienten, die an chronischen Rückenschmerzen oder Depression leiden. Rusu, Pincus und Morley (2012) konnten in einer Studie zeigen, dass Patienten mit Depression und Schmerz einen negativen kognitiven Bias bezüglich gesundheitsbezogener Informationen zeigen. Deshalb ist anzunehmen, dass die Effekte des präsentierten experimentellen Paradigmas in Patientenstichproben größer ausfallen würden und diesen ein stärkerer Negativitätsbias unterliegt. Zusätzlich wäre interessant, das neu entwickelte Stimulusmaterial zu überprüfen und zu validieren, um eine bessere Vergleichbarkeit zwischen den Studien zu erreichen.

Für die klinische Praxis ergeben sich aus den Ergebnissen der Studie 1 verschiedene Implikationen. Die Ergebnisse lassen darauf schließen, dass bereits wenige negative Informationen Auswirkungen auf die Einstellungen gegenüber bestimmten Bewegungen haben können, sodass angenommen werden kann, dass Patienten mit chronischen Rückenschmerzen starke negative Einstellungen haben, die möglicherweise sehr resistent gegenüber Veränderungen sind. Das sollte in der Behandlung berücksichtigt werden, indem über verschiedene Interventionen inhibitorische Lernprozesse angeregt und damit verfestigt werden. Ein Ansatz von Craske und Kollegen (2008) legt den Fokus von Expositionstherapien weg von der Angstreduktion und verstärkt auf die kognitiven Komponenten, nämlich der Erwartungen. Die vorliegenden Ergebnisse unterstützen dies und sollten in der klinischen Praxis zusätzlich berücksichtigt werden. Weiterhin sollte beachtet werden, dass explizite Lernerfahrungen sich von impliziten Einstellungen unterscheiden können („Ich weiß, dass Bewegung mir gut tut und trotzdem schaffe ich es aus irgendeinem Grund nicht.“), sodass in Therapien genauer auf mögliche hemmende Lernfaktoren eingegangen oder psychoedukativ auf mögliche Unterschiede in zugrunde liegenden Einstellungen erklärt werden sollte. Dadurch könnte ambivalentes Verhalten erklären werden und ebenfalls Druck vom Patienten genommen und Misserfolge verringert werden. Psychoedukation könnte auch im Bereich der Prävention wichtig sein.

Da sich Fear-Avoidance Beliefs auch ohne konkretes Bewusstsein entwickeln können, sollten proaktive Maßnahmen getroffen werden und so schon früh einer Chronifizierung entgegenzuwirken.

Die Ergebnisse aus Studie 2 sollten durch eine konfirmatorische Überprüfung der Faktorstruktur und in einer größeren Stichprobe ergänzt werden. In klinischen Studien sollte der Fragebogen eingesetzt werden, um besser zu verstehen, welche Veränderungsprozesse durch eine (erfolgreiche) Therapie angeregt werden. Von besonderem Interesse wäre es verschiedene Therapieverfahren zu vergleichen, um mögliche Wirkmechanismen zu untersuchen. Einer expositionsbasierten Behandlung (Vlaeyen et al., 2002) werden andere Lernprozesse zugeschrieben als beispielsweise einer auf Akzeptanz der Schmerzen basierten Behandlung wie der Acceptance and Commitment Therapy (ACT, Hayes et al. 2006). Beide Therapieverfahren versuchen jedoch mit unterschiedlichen Methoden den Fokus vom Schmerz zu lenken, sodass anzunehmen ist, dass beide Therapieverfahren akkommodative Copingstrategien vermitteln und somit ähnlich wirken. Weiterhin könnte der Fragebogen in Behandlungen, die auf einer Reduktion der Schmerzen abzielen, eingesetzt werden, um zu untersuchen, wie sich diese Behandlungen auf die Wahl der Copingstrategien auswirken. Ein weiterer wichtiger Ansatzpunkt ist zudem die zusätzliche Validierung des Fragebogens in einer Stichprobe bestehend aus Personen mit akuten oder subchronischen Schmerzen, um mehr Wissen über ein günstiges Zusammenspiel assimilativer und akkommodativer Copingstrategien zu erlangen. In der klinischen Praxis könnten daraus Präventions- oder Interventionsprogramme abgeleitet werden, in denen ein unterschiedlich starker Fokus auf den Aufbau assimilativer oder akkommodativer Copingstrategien besteht und somit eine Chronifizierung bzw. die Schwere der Symptomatik reduziert oder unterbunden werden könnte.

Die Ergebnisse der Studie 3 und die weitere Diskussion in Studie 4 zeigen, dass die Forschungslage im Bereich Biofeedback in der Behandlung chronischer Rückenschmerzen mit qualitativ hochwertigen Studien oder bestenfalls mit randomisiert-kontrollierten Studien ergänzt werden sollte. Dabei sollte darauf geachtet werden, dass wichtige Studiencharakteristika, wie die Diagnose, Art und Dauer des Biofeedbacks oder eine genaue Beschreibung der Intervention in der Experimental- sowie Kontrollgruppe, berichtet werden. Dadurch könnte spezifischer untersucht werden, für welche Diagnose welche Biofeedbackmodalität wirksam(er) ist. Dadurch könnten Biofeedbacktherapien optimiert werden. Weiterhin ist unklar, welcher Wirkmechanismus dem Erfolg von Behandlungen mit Biofeedback unterliegt. Daher wären experimentelle Untersuchungen, z.B. Biofeedback mit Placebobedingung (Kapitza et al., 2010), interessant, in denen Moderatoren oder Mediatoren untersucht werden könnten. Gleichzeitig erscheint es sinnvoll eine Verhaltensvariable wie Schmerzverhalten oder Arbeitsunfähigkeit mit zu erheben, um auch Rückschlüsse auf das tatsächliche Verhalten ziehen zu können. Mehr Wissen über die Spezifität von Biofeedback, den

Wirkmechanismen sowie Auswirkungen auf den Alltag könnten in der klinischen Praxis hilfreich sein, um den Einsatz von Biofeedback in der ambulanten oder stationären Versorgung zu erhöhen. Die Ergebnisse der Studie 3 zeigten, dass der Einsatz von Biofeedback sowohl kurz- als auch langfristig wirksam in der Reduktion verschiedener schmerzrelevanter Symptome ist. Somit kann der Einsatz dieser operanten Methode empfohlen werden. Da es sich bei Biofeedback um ein nicht-invasives, verhaltensmedizinisches Verfahren handelt, sollten im Vergleich zur Einnahme von Opioiden weniger Nebenwirkungen auftreten dürfen (Martell et al., 2007). Aus dieser Perspektive sollte zudem untersucht werden, ob der Einsatz von Biofeedback ebenfalls Auswirkungen auf die Anzahl oder Art der Einnahme von Schmerzmedikamenten hat.

5.3 Fazit

In der vorliegenden Dissertation wurden unterschiedliche Lernmechanismen in der Entstehung und Aufrechterhaltung sowie in der Behandlung chronischer Rückenschmerzen untersucht. Für die Entwicklung chronischer Rückenschmerzen werden Fear-Avoidance Beliefs als wichtige Risikofaktoren diskutiert. Durch die Ergebnisse dieser Dissertation konnte erstmals gezeigt werden, dass evaluative Konditionierungsprozesse eine Rolle in der Entstehung von Fear-Avoidance Beliefs spielen. Für die Verbesserung der Behandlung chronischer Rückenschmerzen, steht durch die Entwicklung einer validen, deutschen Version des Pain Solutions Questionnaires zudem ein Fragebogen zur Verfügung, mit dem assimilative und akkommodative Copingstrategien erfasst werden können. Dadurch kann sowohl die Forschung zum Übergang von akuten zu chronischen Schmerzen als auch zu Veränderungsprozessen während Therapien genauer untersucht werden. Im Sinne des „Tailored Treatment“ kann der PaSol zudem eingesetzt werden, um individuell geeignete Interventionen auszuwählen. Für die Behandlung chronischer Rückenschmerzen konnte zudem die kurz- und langfristige Wirksamkeit von Biofeedback als operantes Lernverfahren auf verschiedene schmerzrelevante Zielgrößen nachgewiesen werden. Damit konnte eine Empfehlung für Biofeedback als psychologische Intervention für die Behandlung chronischer Rückenschmerzen ausgesprochen werden.

LITERATURVERZEICHNIS

- Aldrich, S., Eccleston, C., & Crombez, G. (2000). Worrying about chronic pain: vigilance to threat and misdirected problem solving. *Behaviour Research and Therapy*, 38, 457–470.
- Asendorpf, J. B., Conner, M., de Fruyt, F., de Houwer, J., Denissen, J., Fiedler, K., ... Wicherts, J. M. (2012). Recommendations for increasing replicability in psychology. *European Journal of Personality*, 119, 108–119. <https://doi.org/10.1002/per>
- Asmundson, G.J.G., Norton, P.J., Vlaeyen, J. W. S. (2004). Fear-avoidance models of chronic pain: an overview. In G. C. G.J.G. Asmundson, J.W.S. Vlaeyen (Ed.), *Understanding and Treating Fear of Pain* (pp. 3–24). New York: Oxford Press.
- Baeyens, F., Díaz, E., & Ruiz, G. (2005). Resistance to extinction of human evaluative conditioning using a between-subjects design. *Cognition & Emotion*, 19(2), 245–68. <https://doi.org/10.1080/02699930441000300>
- Balagué, F., Mannion, A. F., Pellisé, F., & Cedraschi, C. (2012). Non-specific low back pain. *The Lancet*, 379(9814), 482–491. [https://doi.org/10.1016/S0140-6736\(11\)60610-7](https://doi.org/10.1016/S0140-6736(11)60610-7)
- Barke, A., Baudewig, J., Schmidt-Samoa, C., Dechent, P., & Kröner-Herwig, B. (2012). Neural correlates of fear of movement in high and low fear-avoidant chronic low back pain patients: An event-related fMRI study. *Pain*, 153(3), 540–552. <https://doi.org/10.1016/j.pain.2011.11.012>
- Baumeister, R. F., Bratslavsky, E., Finkenauer, C., & Vohs, K. D. (2001). Bad Is Stronger Than Good. *Review of General Psychology*, 5(4), 323–370. <https://doi.org/10.1037//1089-2680.5.4.323>
- Beaton, D. E., Bombardier, C., Guillemin, F., & Ferraz, M. B. (2000). Guidelines for the process of cross-cultural adaptation of self-report measures. *Spine*, 25(24), 3186–3191. <https://doi.org/10.1097/00007632-200012150-00014>
- Boersma, K., Linton, S., Overmeer, T., Jansson, M., Vlaeyen, J., & De Jong, J. (2004). Lowering fear-avoidance and enhancing function through exposure in vivo: A multiple baseline study across six patients with back pain. *Pain*, 108(1), 8–16. <https://doi.org/10.1016/j.pain.2003.03.001>
- Boos, N., Rieder, R., Schade, V., Spratt, K. F., Semmer, N., & Aebi, M. (1995). The Diagnostic Accuracy of Magnetic Resonance Imaging, Work Perception and Psychosocial Factors in Identifying Symptomatic Disc Herniations. *Spine*, 20(24), 2613–2625.
- Bouton, M. E. (1994). Conditioning , Remembering , and Forgetting. *Journal of Experimental Psychology: Animal Behavior Processes*, 20(3), 219–231.
- Brandtstädter, J. (2007). Hartnäckige Zielverfolgung und flexible Zielanpassung als Entwicklungsressourcen: Das Modell assimilativer und akkommodativer Prozesse. In J. Brandtstädter & U. Lindenberger (Eds.), *Entwicklungspsychologie der Lebensspanne. Ein Lehrbuch*. (pp. 413–445). Stuttgart: Kohlhammer.
- Brandtstädter, J., & Renner, G. (1990). Tenacious goal pursuit and flexible goal adjustment: Explication and age-related analysis of assimilative and accommodative strategies of coping. *Psychology and Aging*, 5(1), 58–67. <https://doi.org/10.1037/0882-7974.5.1.58>
- Breivik, H., Collett, B., Ventafridda, V., Cohen, R., & Gallacher, D. (2006). Survey of chronic pain in Europe: Prevalence, impact on daily life, and treatment. *European Journal of Pain*, 10(4), 287–333. <https://doi.org/10.1016/j.ejpain.2005.06.009>
- Brox, J. I. (2014). Current evidence on catastrophizing and fear avoidance beliefs in low back pain patients. *The Spine Journal*, 14(11), 2679–2681. <https://doi.org/10.1016/j.spinee.2014.08.454>
- Chou, R., & Shekelle, P. (2010). Will this patient develop persistent disabling low back pain? *Jama*, 303(13), 1295–302. <https://doi.org/10.1001/jama.2010.344>
- Craske, M. G., Kircanski, K., Zelikowsky, M., Mystkowski, J., Chowdhury, N., & Baker, A. (2008).

- Optimizing inhibitory learning during exposure therapy. *Behaviour Research and Therapy*, 46(1), 5–27. <https://doi.org/10.1016/j.brat.2007.10.003>
- Craske, M. G., Treanor, M., Conway, C., Zbozinek, T., & Vervliet, B. (2014). Maximizing Exposure Therapy: An Inhibitory Learning Approach. *Behav Res Ther.*, 58, 10–23. <https://doi.org/10.1016/j.brat.2014.04.006>
- Crombez, G., Eccleston, C., Van Hamme, G., & De Vlieger, P. (2008). Attempting to solve the problem of pain: A questionnaire study in acute and chronic pain patients. *Pain*, 137(3), 556–563. <https://doi.org/10.1016/j.pain.2007.10.020>
- Crombez, G., Van Damme, S., & Eccleston, C. (2005). Hypervigilance to pain: An experimental and clinical analysis. *Pain*, 116, 4–7. <https://doi.org/10.1016/j.pain.2005.03.035>
- Crombez, G., Vlaeyen, J. W. S., Heuts, P. H. T. G., & Lysens, R. (1999). Pain-related fear is more disabling than pain itself: Evidence on the role of pain-related fear in chronic back pain disability. *Pain*, 80(1–2), 329–339. [https://doi.org/10.1016/S0304-3959\(98\)00229-2](https://doi.org/10.1016/S0304-3959(98)00229-2)
- De Houwer, J. (2007). A conceptual and theoretical analysis of evaluative conditioning. *The Spanish Journal of Psychology*, 10(2), 230–241. <https://doi.org/psi/11387416/articulos/SJOP0707220230A.PDF>
- De Houwer, J., Thomas, S., & Baeyens, F. (2001). Associative Learning of Likes and Dislikes: A Review of 25 Years of Research on Human Evaluative Conditioning. *Psychological Bulletin*, 127(6), 853–869. <https://doi.org/http://psycnet.apa.org/doi/10.1037/0033-2909.127.6.853>
- De Vlieger, P., Bussche, E. Van Den, Eccleston, C., & Crombez, G. (2006). Finding a solution to the problem of pain: Conceptual formulation and the development of the Pain Solutions Questionnaire (PaSol). *Pain*, 123(3), 285–293. <https://doi.org/10.1016/j.pain.2006.03.005>
- den Hollander, M., de Jong, J. R., Volders, S., Goossens, M. E., Smeets, R. J., & Vlaeyen, J. W. (2010). Fear reduction in patients with chronic pain: a learning theory perspective. *Expert Review of Neurotherapeutics*, 10(11), 1733–1745. <https://doi.org/10.1586/ern.10.115>
- den Hollander, M., de Jong, J. R., Volders, S., Goossens, M. E., Smeets, R. J., Vlaeyen, J. W., ... Vlaeyen, J. W. (2010). Fear reduction in patients with chronic pain: a learning theory perspective. *Expert Review of Neurotherapeutics*, 10(11), 1733–1745. <https://doi.org/10.1586/ern.10.115>
- Etkin, A., & Wager, T. D. (2007). Functional Neuroimaging of Anxiety: A Meta-Analysis of Emotional Processing in PTSD, Social Anxiety Disorder, and Specific Phobia. *Am J Psychiatry*, 164(10), 1476–1488. <https://doi.org/10.1176/appi.ajp.2007.07030504.Functional>
- Flor, H., & Birbaumer, N. (1993). Comparison of the Efficacy of Electromyographic Biofeedback, Cognitive-Behavioral Therapy, and Conservative Medical Interventions in the Treatment of Chronic Musculoskeletal Pain. *Journal of Consulting and Clinical Psychology*. Retrieved from <http://psycnet.apa.org/journals/ccp/61/4/653/>
- Gawronski, B., Gast, A., & De Houwer, J. (2014). Is evaluative conditioning really resistant to extinction? Evidence for changes in evaluative judgements without changes in evaluative representations. *Cognition & Emotion*, 9931(October), 1–15. <https://doi.org/10.1080/02699931.2014.947919>
- Gheldof, E. L. M., de Jong, P. J., Vinck, J., & Houben, R. M. A. (2004). *Attitudes toward physical activity: The role of implicit versus explicit associations*. (G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crombez, Eds.). Oxford: Oxford University Press.
- Glombiewski, J. A., Bernardy, K., & Häuser, W. (2013). Efficacy of EMG- and EEG-Biofeedback in Fibromyalgia Syndrome: A Meta-Analysis and a Systematic Review of Randomized Controlled Trials. *Evidence-Based Complementary and Alternative Medicine*, 2013. <https://doi.org/10.1155/2013/962741>

- Glombiewski, J. A., Hartwich-Tersek, J., & Rief, W. (2010). Two Psychological Interventions Are Effective in Severely Disabled, Chronic Back Pain Patients: A Randomised Controlled Trial. *International Journal of Behavioral Medicine*, 17(2), 97–107. <https://doi.org/10.1007/s12529-009-9070-4>
- Glombiewski, J. A., Riecke, J., Holzapfel, S., Rief, W., König, S., Lachnit, H., & Seifart, U. (2015). Do chronic pain patients show autonomic arousal when confronted with feared movements? An experimental investigation of the fear-avoidance model. *Pain*, 156(3), 547–554. <https://doi.org/10.1097/01.j.pain.0000460329.48633.ce>
- Gore, M., Sadosky, A., Stacey, B. R., Tai, K.-S., & Leslie, D. (2012). The Burden of Chronic Low Back Pain. *Spine*, 37(11), E668–E677. <https://doi.org/10.1097/BRS.0b013e318241e5de>
- Goubert, L., Crombez, G., & De Bourdeaudhuij, I. (2004). Low back pain, disability and back pain myths in a community sample: Prevalence and interrelationships. *European Journal of Pain*, 8(4), 385–394. <https://doi.org/10.1016/j.ejpain.2003.11.004>
- Goubert, L., Crombez, G., Hermans, D., & Vanderstraeten, G. (2003). Implicit attitude towards pictures of back-stressing activities in pain-free subjects and patients with low back pain: An affective priming study. *European Journal of Pain*, 7, 33–42. [https://doi.org/10.1016/S1090-3801\(02\)00054-X](https://doi.org/10.1016/S1090-3801(02)00054-X)
- Goubert, L., Crombez, G., & Peters, M. (2004). Pain-related fear and avoidance: A conditioning perspective. In G. J. G. Asmundson, J. W. S. Vlaeyen, & G. Crombez (Eds.), *Understanding and Treating Fear of Pain* (pp. 25–50). New York: Oxford University Press.
- Goubert, L., Crombez, G., & Van Damme, S. (2004). The role of neuroticism, pain catastrophizing and pain-related fear in vigilance to pain: A structural equations approach. *Pain*, 107(3), 234–241. <https://doi.org/10.1016/j.pain.2003.11.005>
- Greenwald, A., McGhee, D., & Schwartz, J. (1998). Measuring individual differences in implicit cognition: the implicit association test. *J Pers Soc Psychol*, 74, 1464–1480.
- Grotle, M., Vøllestad, N. K., & Brox, J. I. (2006). Clinical course and impact of fear-avoidance beliefs in low back pain: prospective cohort study of acute and chronic low back pain: II. *Spine*, 31(9), 1038–1046. <https://doi.org/10.1097/01.brs.0000214878.01709.0e>
- Grotle, M., Vøllestad, N. K., Veierød, M. B., & Brox, J. I. (2004). Fear-avoidance beliefs and distress in relation to disability in acute and chronic low back pain. *Pain*, 112(3), 343–352. <https://doi.org/10.1016/j.pain.2004.09.020>
- Hasenbring, M., Marienfeld, G., Kuhlendahl, D., & Soyka, D. (1994). Risk factors of chronicity in lumbar disc patients. A prospective investigation of biologic, psychologic, and social predictors of therapy outcome. *Spine*, 19(24), 2759–2765.
- Hayes, S., Luoma, J., Bond, F., Masuda, A., & Lillis, J. (2006). Acceptance and commitment therapy: model, processes and outcomes. *Behav. Res. Ther.*, 44(1), 1–25.
- Helsen, K., Goubert, L., Peters, M. L., & Vlaeyen, J. W. S. (2011). Observational learning and pain-related fear: An experimental study with colored cold pressor tasks. *Journal of Pain*, 12(12), 1230–1239. <https://doi.org/10.1016/j.jpain.2011.07.002>
- Henschke, N., Ostelo, R., van Tulder, M., Vlaeyen, J., Morley, S., Assendelft, W., & Main, C. (2010). Behavioural treatment for chronic low-back pain (Review). *Cochrane Database of Systematic Reviews (Online)*, (7), CD002014. <https://doi.org/10.1002/14651858.CD002014.pub3>
- Henschke, N., Rwigyira, O., Mwambi, V. T., Jwa, V., Morley, S., Wj, A., & Cj, M. (2011). Behavioural treatment for chronic low-back pain (Review), (7).
- Hermans, D., Crombez, G., Vansteenwegen, D., Baeyens, F., & Eelen, P. (2002). Expectancy-learning and evaluative learning in human classical conditioning: Differential effects of extinction.

- Advances in Psychology Research*, Vol. 12, 40, 17–40. [https://doi.org/10.1016/S0005-7967\(01\)00006-7](https://doi.org/10.1016/S0005-7967(01)00006-7)
- Hoffman, B. M., Papas, R. K., Chatkoff, D. K., & Kerns, R. D. (2007). Meta-Analysis of Psychological Interventions for Chronic Low Back Pain. *Health Psychology : Official Journal of the Division of Health Psychology, American Psychological Association*, 26(1), 1–9. <https://doi.org/10.1037/0278-6133.26.1.1>
- Hofmann, S. G., & Smits, J. a J. (2008). Pitfalls of meta-analyses. *The Journal of Nervous and Mental Disease*, 196(9), 716–717. <https://doi.org/10.1097/NMD.0b013e318183fd90>
- Hofmann, W., De Houwer, J., Perugini, M., Baeyens, F., & Crombez, G. (2010). Evaluative conditioning in humans: A meta-analysis. *Psychological Bulletin*, 136(3), 390–421. <https://doi.org/10.1037/a0018916>
- Holzappel, S., Riecke, J., Rief, W., Schneider, J., & Glombiewski, J. A. (2016). Development and Validation of the Behavioral Avoidance Test – Back pain (BAT-Back) for Patients with Chronic Low Back Pain. *The Clinical Journal of Pain*, (JANUARY), 1. <https://doi.org/10.1097/AJP.0000000000000349>
- Jadad, A. R., Moore, R. A., Carroll, D., Jenkinson, C., Reynolds, D. J. M., Gavaghan, D. J., & McQuay, H. J. (1996). Assessing the quality of reports of randomized clinical trials: Is blinding necessary? *Controlled Clinical Trials*, 17(1), 1–12. [https://doi.org/10.1016/0197-2456\(95\)00134-4](https://doi.org/10.1016/0197-2456(95)00134-4)
- Jensen, M. C., Brant-Zawadzki, M. N., Obuchowski, N., Modic, M. T., Malkasian, D., & Ross, J. S. (1994). Magnetic Resonance Imaging of the Lumbar Spine in People without Back Pain. *The New England Journal of Medicine*, 331(2), 69–73.
- Jones, C. R., Olson, M. A., & Fazio, R. H. (2010). Evaluative Conditioning: The “How” Question. *Advances in Experimental Social Psychology*, 43(10), 205–255. [https://doi.org/10.1016/S0065-2601\(10\)43005-1](https://doi.org/10.1016/S0065-2601(10)43005-1)
- Kapitza, K. P., Passie, T., Bernateck, M., & Karst, M. (2010). First non-contingent respiratory biofeedback placebo versus contingent biofeedback in patients with chronic low back pain: A randomized, controlled, double-blind trial. *Applied Psychophysiology Biofeedback*, 35(3), 207–217. <https://doi.org/10.1007/s10484-010-9130-1>
- Kerkhof, I., Vansteenwegen, D., Baeyens, F., & Hermans, D. (2011). Counterconditioning: An effective technique for changing conditioned preferences. *Experimental Psychology*, 58(1), 31–38. <https://doi.org/10.1027/1618-3169/a000063>
- Kugler, K., Wijn, J., Geilen, M., de Jong, J., and Vlaeyen, J. W. S. (1999). The Photograph series of Daily Activities (PHODA). Institute for Rehabilitation Research and School for Physiotherapy Heerlen, The Netherlands.
- Lang, P.J., Bradley, M.M., & Cuthbert, B. N. (2008). *International affective picture system (IAPS): Affective ratings of pictures and instruction manual. Technical Report A-8. Technical Report A-8.* University of Florida. Retrieved from [ftp://dhcp-129-105-171-164.psych.northwestern.edu/OpenShare/ESPN/IAPS 1-16/IAPS 1-16/IAPSmanual.pdf](ftp://dhcp-129-105-171-164.psych.northwestern.edu/OpenShare/ESPN/IAPS%201-16/IAPS%201-16/IAPSmanual.pdf)
- Leeuw, M., Goossens, M. E. J. B., Linton, S. J., Crombez, G., Boersma, K., & Vlaeyen, J. W. S. (2007). The Fear-Avoidance Model of Musculoskeletal Pain: Current State of Scientific Evidence. *Journal of Behavioral Medicine*, 30(1), 77–94. <https://doi.org/10.1007/s10865-006-9085-0>
- Leeuw, M., Goossens, M., van Breukelen, G., de Jong, J., Heuts, P., Smeets, R., ... Vlaeyen, J. (2008). Exposure in vivo versus operant graded activity in chronic low back pain patients: Results of a randomized controlled trial. *Pain*, 138, 192–207.
- Linton, S. J. (2000). A review of psychological risk factors in back and neck pain. *Spine*, 25(9), 1148–1156. <https://doi.org/10.1097/00007632-200005010-00017>

- Linton, S. J., Buer, N., Vlaeyen, J., & Hellsing, A.-L. (2000). Are fear-avoidance beliefs related to the inception of an episode of back pain? A prospective study. *Psychology & Health*, 14(6), 1051–1059. <https://doi.org/10.1080/08870440008407366>
- Louw, Q. A., Morris, L. D., & Grimmer-Somers, K. (2007). The prevalence of low back pain in Africa: a systematic review. *BMC Musculoskelet Disord*, 8, 105. <https://doi.org/10.1186/1471-2474-8-105>
- Magnusson, M. L., Chow, D. H., Diamandopoulos, Z., & Pope, M. H. (2008). Motor Control Learning in Chronic Low Back Pain. *Spine (Phila Pa 1976)*, 33(16), E532-8. <https://doi.org/10.1097/BRS.0b013e31817dfd9a>
- Maniadakis, N., & Gray, A. (2000). The economic burden of back pain in the UK. *Pain*, 84(1), 95–103. [https://doi.org/10.1016/S0304-3959\(99\)00187-6](https://doi.org/10.1016/S0304-3959(99)00187-6)
- Martell, B. A., O'Connor, P. G., Kerns, R. D., Becker, W. C., Morales, K. H., Kosten, T. R., & Fiellin, D. A. (2007). Systematic review: Opioid treatment for chronic back pain: Prevalence, efficacy, and association with addiction. *Annals of Internal Medicine*, 146(2), 116–127. <https://doi.org/10.7326/0003-4819-146-2-200701160-00006>
- Martin, I., & Levey, A. B. (1978). EVALUATIVE CONDITIONING. *Adv. Behav. Res. Ther.*, 1, 57–102.
- McCracken, L. M., Carson, J. W., Eccleston, C., & Keefe, F. J. (2004). Acceptance and change in the context of chronic pain. *Pain*, 109(1–2), 4–7. <https://doi.org/10.1016/j.pain.2004.02.006>
- McCracken, L. M., Vowles, K. E., & Eccleston, C. (2004). Acceptance of chronic pain: Component analysis and a revised assessment method. *Pain*, 107(1–2), 159–166. <https://doi.org/10.1016/j.pain.2003.10.012>
- McCracken, L., Vowles, K., & Eccleston, C. (2004). Acceptance of chronic pain: component analysis and a revised assessment method. *Pain*, 107(1–2), 159–166.
- Meulders, A., & Vansteenwegen, Debora; Vlaeyen, J. W. S. (2011). The acquisition of fear of movement-related pain and associative learning: A novel pain-relevant human fear conditioning paradigm. *Pain*, 152(11), 2460–2469. <https://doi.org/10.1016/j.pain.2011.05.015>
- Morley, S. (2011). Efficacy and effectiveness of cognitive behaviour therapy for chronic pain: Progress and some challenges. *Pain*, 152(SUPPL.3), S99–S106. <https://doi.org/10.1016/j.pain.2010.10.042>
- Neblett, R. (2016). Surface Electromyographic (SEMG) Biofeedback for Chronic Low Back Pain. *Healthcare*, 4(2), 27. <https://doi.org/10.3390/healthcare4020027>
- Nestoriuc, Y., & Martin, A. (2007). Efficacy of biofeedback for migraine: A meta-analysis. *Pain*, 128(1–2), 111–127. <https://doi.org/10.1016/j.pain.2006.09.007>
- Nestoriuc, Y., Rief, W., & Martin, A. (2008). Meta-Analysis of Biofeedback for Tension-Type Headache: Efficacy, Specificity, and Treatment Moderators. *Journal of Consulting and Clinical Psychology*, 76(3), 379–396. <https://doi.org/10.1037/0022-006X.76.3.379>
- Nouwen, A. (1983). EMG biofeedback used to reduce standing levels of paraspinal muscle tension in chronic low back pain. *Pain*, 17(4), 353–360. [https://doi.org/10.1016/0304-3959\(83\)90166-5](https://doi.org/10.1016/0304-3959(83)90166-5)
- Pellisé, F., Balagué, F., Rajmil, L., Cedraschi, C., Aguirre, M., Fontecha, C. G., ... Ferrer, M. (2009). Prevalence of low back pain and its effect on health-related quality of life in adolescents. *Archives of Pediatrics and Adolescent Medicine*, 163(1), 65–71. Retrieved from <http://www.scopus.com/inward/record.url?eid=2-s2.0-61449183606&partnerID=40&md5=fe929164878573446aa6500c249a5bad>
- Pfingsten, M., & Hildebrandt, J. (2011). Rückenschmerzen. In B. Kröner-Herwig, J. Frettlöh, R. Klinger, & P. Nilges (Eds.), *Schmerzpsychotherapie. Grundlagen - Diagnostik - Krankheitsbilder - Behandlung* (7., pp. 431–452). Berlin Heidelberg: Springer-Verlag. <https://doi.org/10.1007/978->

3-642-12783-0

- Pincus, T., Burton, a K., Vogel, S., & Field, A. P. (2002). A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine (Phila Pa 1976)*, 27(5), E109-20. <https://doi.org/10.1097/00007632-200203010-00017>
- Pincus, T., Smeets, R. J. E. M., Simmonds, M. J., & Sullivan, M. J. L. (2010). The fear avoidance model disentangled: improving the clinical utility of the fear avoidance model. *The Clinical Journal of Pain*, 26(9), 739–746. <https://doi.org/10.1097/AJP.0b013e3181f15d45>
- Raes, A. K., & De Raedt, R. (2012). The Effect of Counterconditioning on Evaluative Responses and Harm Expectancy in a Fear Conditioning Paradigm. *Behavior Therapy*, 43(4), 757–767. <https://doi.org/10.1016/j.beth.2012.03.012>
- Reinecker, H. (2005). Psychologische Grundlagen der Verhaltenstherapie. In H. Reinecker (Ed.), *Grundlagen der Verhaltenstherapie*. (3rd ed., pp. 74–123). Weinheim: Beltz PVU.
- Rescorla, R. a. (1966). Predictability and the number of pairings in Pavlovian fear conditioning. *Psychonomic Science*, 4, 383–384.
- Rescorla, R. a. (1988). Pavlovian conditioning. It's not what you think it is. *The American Psychologist*, 43(3), 151–160. <https://doi.org/10.1037/0003-066X.43.3.151>
- Rief, W., & Birbaumer, N. (2011). Grundsätzliches zu Biofeedback. In W. Rief & N. Birbaumer (Eds.), *Biofeedback. Grundlagen - Indikationen - Kommunikation - Vorgehen*. (3., pp. 1–7). Stuttgart: Schattauer.
- Rief, W., & Glombiewski, J. A. (2016). Expectation-Focused Psychological Interventions (EFPI). *Verhaltenstherapie*, 47–54. <https://doi.org/10.1159/000442374>
- Rief, W., Glombiewski, J. a, Gollwitzer, M., Schubö, A., Schwarting, R., & Thorwart, A. (2015). Expectancies as core features of mental disorders. *Current Opinion in Psychiatry*, 28(5), 378–85. <https://doi.org/10.1097/YCO.0000000000000184>
- Rosa Esteve, M., & Camacho, L. (2008). Anxiety sensitivity, body vigilance and fear of pain. *Behaviour Research and Therapy*, 46(6), 715–727. <https://doi.org/10.1016/j.brat.2008.02.012>
- Rosenstiel, a K., & Keefe, F. J. (1983). The use of coping strategies in chronic low back pain patients: relationship to patient characteristics and current adjustment. *Pain*, 17(1), 33–44. [https://doi.org/10.1016/0304-3959\(83\)90125-2](https://doi.org/10.1016/0304-3959(83)90125-2)
- Rozin, P., & Royzman, E. B. (2001). Negativity Bias, Negativity Dominance, and Contagion Paul. *Personality and Social Psychology Review*, 5(4), 296–320. <https://doi.org/10.1207/S15327957PSPR0504>
- Rusu, A. C., Pincus, T., & Morley, S. (2012). Depressed pain patients differ from other depressed groups: Examination of cognitive content in a sentence completion task. *Pain*, 153(9), 1898–1904. <https://doi.org/10.1016/j.pain.2012.05.034>
- Sanders, S. H. (2002). Operant conditioning with chronic pain: back to basics. In D. Turk & R. Gatchel (Eds.), *Psychological approaches to pain management*. New York: The Guilford Press.
- Santaella da Fonseca Lopes de Sousa, K., Garcia Orfale, A., Mara Meireles, S., Roberto Leite, J., & Natour, J. (2009). Assessment of a Biofeedback Program to Treat Chronic Low Back Pain. *Journal of Musculoskeletal Pain*, 17(4), 369–377. <https://doi.org/10.3109/10582450903284828>
- Schmitz, U., Saile, H., & Nilges, P. (1996). Coping with chronic pain: Flexible goal adjustment as an interactive buffer against pain-related distress. *Pain*, 67(1), 41–51. [https://doi.org/10.1016/0304-3959\(96\)03108-9](https://doi.org/10.1016/0304-3959(96)03108-9)
- Scholich, S. L., Hallner, D., Wittenberg, R. H., Hasenbring, M. I., & Rusu, A. C. (2012). The relationship between pain, disability, quality of life and cognitive-behavioural factors in chronic back pain. *Disability and Rehabilitation*, 34(23), 1993–2000.

- <https://doi.org/10.3109/09638288.2012.667187>
- Schwartz, N. M., & Schwartz, M. S. (2003). Definitions of Biofeedback and Applied Psychophysiology. In M. S. Schwartz & F. Andrasik (Eds.), *Biofeedback: A practitioner's guide* (3., pp. 27–42). New York: Guilford Press.
- Sieben, J. M., Vlaeyen, J. W. S., Portegijs, P. J. M., Verbunt, J. A., Van Riet-Rutgers, S., Kester, A. D. M., ... André Knottnerus, J. (2005). A longitudinal study on the predictive validity of the fear-avoidance model in low back pain. *Pain*, 117(1–2), 162–170.
<https://doi.org/10.1016/j.pain.2005.06.002>
- Sielski, R., Rief, W., & Glombiewski, J. A. (2016). Efficacy of Biofeedback in Chronic back Pain: a Meta-Analysis. *International Journal of Behavioral Medicine*. <https://doi.org/10.1007/s12529-016-9572-9>
- Stavemann, H. H. (2008). Der typische Ablauf ambulanter KVT. In H. H. Stavemann (Ed.), *KVT-Praxis. Strategien und Leitfäden für die Kognitive Verhaltenstherapie*. (pp. 17–271). Weinheim: Beltz Verlag.
- Stuckey, S. J., Jacobs, A., & Goldfarb, J. (1986). EMG biofeedback training, relaxation training, and placebo for the relief of chronic back pain. *Perceptual and Motor Skills*, 63(3), 1023–1036.
<https://doi.org/10.2466/pms.1986.63.3.1023>
- Turk, D. C. (2002). Clinical effectiveness and cost-effectiveness of treatments for patients with chronic pain. *Clin J Pain*, 18(6), 355–365. <https://doi.org/10.1097/00002508-200211000-00003>
- Van Damme, S., Crombez, G., & Eccleston, C. (2002). Retarded disengagement from pain cues : the effects of pain catastrophizing and pain expectancy. *Pain*, 100, 111–118.
- Van Damme, S., Crombez, G., & Eccleston, C. (2008). Coping with pain: A motivational perspective. *Pain*, 139(1), 1–4. <https://doi.org/10.1016/j.pain.2008.07.022>
- Verbunt, J. A., Seelen, H. A., Vlaeyen, J. W., Van der Heijden, G. J., & Knottnerus, J. A. (2003). Fear of injury and physical deconditioning in patients with chronic low back pain. *Archives of Physical Medicine and Rehabilitation*, 84(8), 1227–1232. [https://doi.org/10.1016/S0003-9993\(03\)00132-1](https://doi.org/10.1016/S0003-9993(03)00132-1)
- Vlaeyen, J., Jong, J., Geilen, M., Heuts, P., & van Breukelen, G. (2002). The Treatment of Fear of Movement/(Re)injury in Chronic Low Back Pain: Further Evidence on the Effectiveness of Exposure In Vivo. *The Clinical Journal of Pain*, 18, 251–261. <https://doi.org/10.1097/00002508-200207000-00006>
- Vlaeyen, J., Morley, S., Linton, S., Boersma, K., & de Jong, J. (2012). Essential Guide to Treatment. In J. Vlaeyen, S. Morley, S. Linton, K. Boersma, & J. de Jong (Eds.), *Pain-Related Fear: Exposure-Based Treatment for Chronic Pain* (pp. 67–94). Seattle: IASP.
- Vlaeyen, J. W. S., De Jong, J., Geilen, M., Heuts, P. H. T. G., & Van Breukelen, G. (2001). Graded exposure in vivo in the treatment of pain-related fear: A replicated single-case experimental design in four patients with chronic low back pain. *Behaviour Research and Therapy*, 39(2), 151–166. [https://doi.org/10.1016/S0005-7967\(99\)00174-6](https://doi.org/10.1016/S0005-7967(99)00174-6)
- Vlaeyen, J. W. S., De Jong, J. R., Onghena, P., Kerckhoffs-Hanssen, M., & Kole-Snijders, A. M. J. (2002). Can pain-related fear be reduced? The application of cognitive-behavioural exposure in vivo. *Pain Research and Management*, 7(3), 144–153. <https://doi.org/10.1155/2002/493463>
- Vlaeyen, J. W. S., & Linton, S. J. (2000). Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain*, 85(3), 317–332. [https://doi.org/10.1016/S0304-3959\(99\)00242-0](https://doi.org/10.1016/S0304-3959(99)00242-0)
- Vlaeyen, J. W. S., Morley, S. J., Linton, S. J., Boersma, K., & de Jong, J. (2012). The Context of the Fear-Avoidance Model. In *Pain-Related Fear. Exposure-Based Treatment for Chronic Pain* (pp. 1–24).

- Walther, E., Nagengast, B., & Trasselli, C. (2005). Evaluative conditioning in social psychology: Facts and speculations. *Cognition & Emotion*, 19(2), 175–96.
<https://doi.org/10.1080/02699930441000274>
- Wenig, C. M., Schmidt, C. O., Kohlmann, T., & Schweikert, B. (2009). Costs of back pain in Germany. *European Journal of Pain (London, England)*, 13(3), 280–6.
<https://doi.org/10.1016/j.ejpain.2008.04.005>
- Wertli, M. M., Rasmussen-Barr, E., Weiser, S., Bachmann, L. M., & Brunner, F. (2014). The role of fear avoidance beliefs as a prognostic factor for outcome in patients with nonspecific low back pain: a systematic review. *The Spine Journal*, 14(5), 816–36.e4.
<https://doi.org/10.1016/j.spinee.2013.09.036>
- Woby, S. R., Watson, P. J., Roach, N. K., & Urmston, M. (2004). Are changes in fear-avoidance beliefs, catastrophizing, and appraisals of control, predictive of changes in chronic low back pain and disability? *European Journal of Pain*, 8(3), 201–210.
<https://doi.org/10.1016/j.ejpain.2003.08.002>
- Woods, M. P., & Asmundson, G. J. G. (2008). Evaluating the efficacy of graded in vivo exposure for the treatment of fear in patients with chronic back pain: A randomized controlled clinical trial. *Pain*, 136(3), 271–280. <https://doi.org/10.1016/j.pain.2007.06.037>
- Wrosch, C., Scheier, M. F., Miller, G. E., Schulz, R., & Carver, C. S. (2003). Adaptive Self-Regulation of Unattainable Goals: Goal Disengagement, Goal Reengagement, and Subjective Well-Being. *Personality and Social Psychology Bulletin*, 29(12), 1494–1508.
<https://doi.org/10.1177/0146167203256921>

APPENDIX

A: Studien

A.1 Studie 1

Forming attitudes toward movements:

The role of evaluative conditioning in the acquisition of fear-avoidance beliefs.

Robert Sielski¹, Sara Lucke², Metin Uengoer², & Julia A. Glombiewski¹

¹Department for Clinical Psychology and Psychotherapy, University of Marburg

²Department for Associative Learning, University of Marburg

Corresponding author

Robert Sielski
Philipps-University of Marburg
Department for Clinical Psychology and Psychotherapy
Gutenbergstraße 18
35032 Marburg, Germany
Email: robert.sielski@staff.uni-marburg.de

Acknowledgments

The study was supported by a doctoral thesis scholarship from the University of Marburg.

Conflicts of interest

The authors report no conflicts of interest.

Abstract

Fear-avoidance beliefs (FAB) are important in the development and maintenance of chronic low back pain (CLBP). Although fear-avoidance is often conceptualized as a conditioned fear response explained via classical and operant conditioning, evidence for the presence of fear is inconsistent. In the absence of fear, another potential learning mechanism is evaluative conditioning (EC). The objectives of this study were to better understand the development and modification of FAB by investigating whether EC affects attitudes towards back-stressing movements among healthy individuals. Using an EC paradigm, pictures showing back-stressing movements were paired with affectively positive or negative unconditioned stimuli (US). Changes in attitudes were tested implicitly using an Affective Priming Task and explicitly using VASs for valence, harmfulness, and pain. In a counter-conditioning phase, pictures of movements were paired with US of the opposite valence.

In study 1, among 33 participants, the affective priming data indicated a negative shift in implicit evaluative responses towards back-stressing movements. Explicit attitudes data indicated that participants rated the movements as more painful after EC. No change in the positive direction occurred.

In study 2, among 50 participants, no implicit affective priming effect was found. Explicit attitudes data again indicated that participants rated the movements as more negatively valenced and more painful after EC. Again, no change in the positive direction occurred.

In conclusion, both experiments suggest that EC can change attitudes towards back-stressing movements in the negative direction on explicit (and implicit) levels. Implications for the prevention of FAB and the treatment of CLBP are discussed.

Key words: Fear-avoidance beliefs; Evaluative Conditioning; Affective Priming Task; Experimental Paradigm

Introduction

The fear-avoidance model is the most influential model of the development and maintenance of chronic low back pain (CLBP) [9,52].

The core of the fear-avoidance model is fear of movement, which is sometimes conceptualized as a conditioned fear response to an anticipated movement [12,35] and sometimes as fear-avoidance beliefs (FAB) [58] or both [15,41]. When conceptualized as a conditioned fear response, classical conditioning – especially expectancy learning – and operant conditioning are assumed to be the main mechanisms [25,52]: according to Vlaeyen and Linton (2000) [52], the association between an unconditioned stimulus (US; pain) and a neutral stimulus (NS; back-stressing movement) can form a conditioned stimulus (CS; back-stressing movement) and leads to the conditioned reaction (CR; fear), resulting in avoidance of the CS (back stressing movement) [35,39,53]. Observing another person experiencing back pain while performing a back-stressing movement can also lead to acquisition of fear of that movement [16,19].

However, research on fear responses among CLBP patients provides inconsistent evidence for the presence of conditioned fear reactions [3,12,41]. Several authors [3,41] argue that fear of movement should instead be conceptualized as health-related beliefs, such as FAB [58].

FAB are described as negative attitudes towards back-stressing movements that lead to avoidance behavior [7], p. 486). FAB are found not only among individuals with CLBP, but also among many pain-free individuals as well as health care providers [7,13,14,28]. FAB can trigger pain-related fear in the presence of acute pain and can lead to delayed recovery or development of chronic pain [14,17,33,57].

Knowledge on how FAB develop and subside is crucial for prevention and treatment of CLBP. Findings from research on fear acquisition as a conditioned reaction to movement may not fully apply to FAB. A review of research on attitude formation suggests evaluative conditioning (EC) as an additional learning mechanism that may influence the development of FAB.

Social psychological research indicates that attitudes can be formed through EC. EC refers to the transfer of valence from positive or negative affective stimuli (US) to neutral stimuli (which become CS) due to their co-occurrence. Unlike other forms of conditioning, EC mostly affects the change in the evaluative response [23]. Additionally, EC effects seem to be resistant to extinction and can develop without CS-US contingency [11,27]. These attitudes may be evident both at the level of explicit awareness and at an implicit, unconscious level, may be activated automatically once acquired, and can impact behavior [20,30].

The primary aim of the present series of experiments was to investigate whether an EC paradigm can form or change attitudes towards back-stressing movements among healthy individuals in either a negative, fearful-avoidant manner, or in a positive, protective manner.

To our knowledge, this is the first study examining evaluative conditioning as a learning mechanism underlying the acquisition of fear-avoidance beliefs.

Methods

Pilot Study

Prior to the experiments, a pilot study examined the adequacy of the experimental stimuli, especially the neutral/conditioned stimuli (CS). In an attempt to enhance ecological validity, pictures from the Photograph Series of Daily Activities (PHODA; [31]) were used as CS. The PHODA consists of 100 pictures of daily activities and has been used as a diagnostic and measurement instrument to assess beliefs about the harmfulness of specific movements [24,50,51]. First, $N = 74$ healthy students ($M = 22.72$ years, $SD = 2.77$; 70.3% female) rated the PHODA pictures on the dimensions of harmfulness and pain on a scale from 0 to 10, ranging from not at all harmful/painful to very harmful/painful (online assessment). These student ratings were compared to PHODA ratings from a prior study of CLBP patients [12]. PHODA pictures were selected as appropriate CS when 1) students' ratings were neutral (thus, learning could occur in either a positive or negative direction), and 2) patients perceived the movements as harmful (thus, movements were relevant for a sample with CLBP). Next, the selected pictures were used in a pilot affective priming paradigm with a sample of $N = 37$ healthy students ($M = 22.70$ years, $SD = 2.93$; 67.6% female). The results showed no differences between PHODA pictures paired with negative US and PHODA pictures paired with positive US. Thus, it was concluded that no learning occurred during the acquisition phase. Potential explanations for these results were discussed within the pain research group and associative learning group of the department of psychology at the Philipps-University Marburg. Additionally, a panel of experts in the field of pain-related fear and experimental research in chronic pain was consulted for further input. Study participants' feedback was also integrated in these discussions: some participants reported that the pictures were out-of-date with respect to clothing style or home furnishings (if shown) and therefore were perceived as "funny." Further consideration of the pictures also suggested that they might be too dark and low-contrast, especially for subliminal presentation in an affective priming paradigm. Participants may also have been confused by the complexity of the pictures, e.g. inclusion of different (partly unrelated) objects, which may have made it unclear what they should rate.

Therefore, we decided to develop simplistic stimuli adapted for the purposes of our study, i.e. pictures of movements that are performed in everyday life and potentially elicit fear or are perceived

as harmful in patients with CLBP. Based on these criteria, three categories of movements were chosen, namely: 1) bending, 2) lifting (one-sided), and 3) moving (e.g., jumping). These categories have been previously found to be associated with fear of harm from movements, subjective disability, and avoidance behavior and were closely related to the movements shown in the PHODA pictures described above [26,44]. Seven different movements were photographed and then transformed into black silhouettes of a man or a woman on a white background. Thus, in these pictures the movement should be clear to the participant and no other information is given about the context or person.

Study 1

Participants

The sample consisted of 33 students (26 female, 7 male) from the Philipps-University Marburg, who received course credit for participation in the study. The mean age was 22.91 years ($SD = 4.67$; range: 18-42). 73% of the participants were studying Psychology as a major.

All participants gave informed consent and none were aware of the research hypotheses. This study was approved by the Ethics Committee of the Faculty of Psychology of the Philipps-University Marburg.

Assessment of implicit and explicit learning effects

Implicit assessment of attitudes: Affective Priming Task (APT)

The Affective Priming Task (APT) is frequently used to examine implicit attitudes. During the APT, participants are instructed to evaluate the valence of a target, e.g., a word. The targets are preceded by positive, negative, or neutral primes. The duration of prime presentation is very short and participants are told to focus on correctly evaluating the target as quickly as possible. Prior research has shown that targets with the same valence as the preceding primes (positive-positive, negative-negative) are evaluated faster (i.e., reaction time is shorter) than incongruent prime-target combinations (positive-negative, negative-positive) [20,22]. These results suggest the automatic activation of attitudes through the short presentation of a negatively or positively valenced stimulus.

Explicit assessment of attitudes: CS ratings

Visual analogue scales (VASs) for the dimensions of valence (positive vs. negative), harmfulness (not at all harmful vs. very harmful), and pain (not at all painful vs. very painful) were used to assess explicit attitudes toward back-stressing movements at baseline and attitude changes over the course of the study. The scales ranged from 0 to 100.

Procedure

The study consisted of two phases. In the first phase, the *stimulus selection phase*, baseline data was collected one week prior to the *experimental phase* to select neutrally rated stimuli. The structure of the study was described in advance and participants were told that the study was about reaction time in tasks with visual stimuli; no information about hypotheses was provided. Informed consent was obtained prior to the *stimulus selection phase*. The procedure is shown in figure 1.

Stimulus selection phase. Baseline ratings were assessed online using the software Enterprise Feedback Suite (EFS) Survey. This part of the study could be completed from the participant's home computer. The pictures (CS) were presented against a light grey background, as 600x800 resolution image files. Participants were asked to rate the movements shown on the dimensions of valence, harmfulness and pain. Ratings for harmfulness and pain were assessed to account for the complexity of attitudes potentially related to fear-avoidance beliefs. The VASs for each dimension were placed underneath the picture and were always in the same order. The presentation order of the stimuli was randomized among participants.

After two practice trials, participants rated 14 pictures showing seven different movements, each performed by a silhouette of a man and a woman. Participants were instructed to rely on their first, spontaneous impression when completing the VASs and were told that there were no right or wrong answers.

Next, participants completed the following questionnaires: the German version of the Tampa Scale for Kinesiophobia (TSK; [37,46], the Pain Catastrophizing Scale (PCS; [36,48], the Fear-Avoidance Beliefs Questionnaire (FABQ; [55]), the Pain and Vigilance Questionnaire [34], and the General Depression Scale (GDS, "Allgemeine Depressionsskala"; [18]). At the end of the stimulus selection phase, demographic variables (e.g., sex, age) were assessed.

Experimental phase. The experimental part of the study took place in a sound-attenuated, air-conditioned laboratory with a steady temperature of 22°C and steady fluorescent lighting. All phases were performed on a 60Hz 16" CRT-computer monitor placed on a table at a stable height of 83cm. Participants were seated approximately 50cm in front of the monitor. All pictures were presented 15cm by 20cm against a black background, as 600x800 resolution image files. Targets were presented in the middle of the screen. Reaction times in the APTs were registered by a manual switch with two buttons, which was attached to the table in front of the monitor. Participants were instructed to use their left index finger for the left button and their right index finger for the right button. Explicit ratings were assessed using a VAS from 0 to 100 for valence, harmfulness, and pain, as assessed at baseline. Participants moved and clicked a mouse to complete the VASs. The

presentation of stimuli and data collection was performed by Presentation® software (Version 14.0, www.neurobs.com).

[Insert figure 1 about here]

Stimulus selection and timing of trials in the EC phase

Conditioned stimuli (CS). As described above, the pictures used as CS in the Evaluative Conditioning (EC) and Counter Conditioning (CC) phases were developed specifically for this study (see appendix A “Conditioned Stimuli”). CS paired with a negative US in the EC phase and with a positive US in the CC phase will be referred to as CS-neg/pos. CS paired with a positive US in the EC phase and with a negative US in the CC phase will be referred to as CS-pos/neg. Two of the seven movements were used as CS-neg/pos or CS-pos/neg, respectively. The order of the conditioning, i.e. whether positive or negative in the EC phase and negative or positive in the CC phase consequently, for the two movements was counterbalanced across participants. The pictures used as CS showed a person bending over and a person wiping the floor. Each movement was shown using male and female silhouettes and the direction of the movement (to the left or right) was varied. These pictures were chosen because the ratings (aggregated for sex and direction) were considered as neutral¹ on all three dimensions: valence ($M_{\text{bending}} = 52.55$, $SD = 14.47$; $M_{\text{wiping}} = 50.55$, $SD = 15.55$), harmfulness ($M_{\text{bending}} = 50.33$, $SD = 19.55$; $M_{\text{wiping}} = 45.73$, $SD = 18.67$), and expected pain ($M_{\text{bending}} = 45.14$, $SD = 20.78$; $M_{\text{wiping}} = 43.15$, $SD = 21.25$) at baseline by the participants. There was no difference between the selected movements (bending vs. wiping) at baseline for any dimension, valence: $t(32) = -0.92$, $p = .365$; harmfulness: $t(32) = -1.62$, $p = .116$; pain: $t(32) = -0.55$, $p = .584$.

Unconditioned stimuli (US): Eight pictures² from the International Affective Picture System (IAPS) [32] were used as US. Four pictures had a positive valence (e.g., No. 2347, children smiling) and four pictures had a negative valence (e.g., No. 3220, man lying in a hospital bed). Selection criteria for US, that were used in the experiment, were high positive and negative ratings for valence ($M_{\text{positive}} = 7.53$; $M_{\text{negative}} = 2.95$), $t(3) = 51.43$, $p < .01$, and similar high ratings for arousal ($M_{\text{positive}} = 5.30$; $M_{\text{negative}} = 5.45$), $t(3) = -0.50$, $p = .650$, on 9-point-scales ranging from 1 to 9 with higher values indicating more positive valences and higher arousal. Ratings were taken from the instruction manual for the IAPS [32]. Affective extremity, as measured by the absolute value of deviation from the mean, was similar across positive and negative pictures selected ($M_{\text{positive}} = 2.53$; $M_{\text{negative}} = 2.10$), $t(3) = 1.72$, $p = .184$).

¹ A mean close to 50 on a scale from 0 to 100 was considered as neutral, with higher values indicating more negative, more harmful, and more painful ratings.

² Positive US: 2347, 8461, 8496, 8540; Negative US: 2457, 3220, 8230, 9599

Stimulus selection

Primes. The CS from the EC and CC phases were used as primes for the APTs. The CS-neg/pos were expected to be negative primes and the CS-pos/neg were expected to be positive primes in the first APT, since the CS-neg/pos were paired with negative US and the CS-pos/neg were paired with positive US in the EC phase. After the CC phase, due to the reversed CS-US contingency the primes were expected to be neutral in the second APT.

Targets. 48 German words selected from “The Berlin Affective Word List Reloaded” (BAWL-R) [54] were used as targets in the APTs (see appendix B “List of target words”). The words were divided into two positive sets and two negative sets. Each set consisted of six nouns and six adjectives. Next, two word lists were compiled, each with one positive and one negative set of words. One word list was used for APT-1 (= after the EC phase) and the other was used for APT-2 (= after the CC phase). The order of the word lists was counterbalanced across participants. The word lists were matched for word length ($M = 6$ characters; $SD = 0.72$), $t(11) = 0$, $p = 1$, and for affective extremity, $t(11) = 0.54$, $p = .603$. Affective valence differed significantly between the positive and negative words ($M_{\text{positive}} = 2.39$; $M_{\text{negative}} = -2.36$) on a 7-point-scale ranging from -3 (very negative) to +3 (very positive). There were also significant differences on ratings for affective arousal, with negative words showing higher arousal values ($M = 3.90$; $SD = 0.45$) than positive words ($M = 2.75$; $SD = 0.57$) on a 5-point-scale ranging from 1 (low arousal) to 5 (high arousal), $t(11) = -6.75$, $p < .001$. It was necessary to accept the differences in affective arousal in order to select word sets that matched for word length and affective extremity, as prior research has shown that relative to positive words, negative words elicit higher ratings for arousal even when the absolute value of the valence rating is similar [29]. In addition to the norms reported by the developers of the BAWL-R, the affective valence of the words was confirmed in a pilot study conducted by the current authors in a healthy student sample.

Study design

Between the *stimulus selection phase* and the *experimental phase* in the laboratory, stimuli were selected based on ratings at baseline. Four pictures showing two different movements with neutral ratings were selected as stimuli for acquisition during the EC phase and for extinction during the CC phase and as primes for the testing phases. Two pictures showing the same movement were chosen as CS-neg/pos, and the other two pictures were chosen as CS-pos/neg. The allocation of movements to CS-neg/pos and CS-pos/neg was counterbalanced across participants.

The *experimental phase* consisted of four phases: EC phase, first testing phase (implicit: APT-1; explicit: CS ratings), CC phase, second testing phase (implicit: APT-2; explicit: CS ratings). Instructions

were provided on the monitor and participants had the opportunity to ask questions before beginning any task.

Design and timing of trials

In the EC phase, participants were told that they would be shown several pictures and that they should look attentively and memorize the pictures due to their importance for the subsequent phases. During the EC phase, participants completed 40 trials, subdivided into five blocks of eight trials. The presentation order of the stimuli was randomized within each block. Each trial began with a fixation cross for 1000ms. Immediately afterwards, a CS was presented for 1000ms followed by a US for 1000ms. The inter-trial interval was 4000ms. Participants were given the opportunity to take a brief break after each block. They could begin the next block independently by pushing the spacebar.

After the EC phase, the first testing phase began with APT-1. In APT-1, all CS (CS-neg/pos; CS-pos/neg) were used as primes and words from one of the two 24-word lists (12 positive, 12 negative) from the BAWL-R were used as targets. Every prime was paired with every positive and negative word in the course of APT-1 and this phase was subdivided into six blocks with 16 trials each, resulting in 96 total trials. Each prime was presented four times in random order within each block, twice with a positive target and twice with a negative target. Accordingly, each block consisted of eight congruent and eight incongruent trials. Prior to the first block, participants were told that pairs of pictures and words would be presented on the screen. They were instructed to attend to the word and to evaluate the valence of the presented word correctly and as quickly as possible by pressing “POSITIVE” or “NEGATIVE” with the right or left button on the manual switch. The assignment of “positive” and “negative” to the left vs. right button was counterbalanced across participants. There were ten practice trials prior to the first block. Primes in the practice trials were black-and-white pictures unrelated to the CS. Practice targets were chosen from the BAWL-R, but were not used for the actual trials of APT-1 or APT-2. In the practice trials only, feedback was given after every trial, and after ten practice trials, the percentage of correct answers and omissions was displayed. Participants had to provide correct answers on 80% of practice trials to proceed, otherwise the practice trials were repeated. Each trial began with a fixation cross for 1000ms, followed by a prime for 200ms and a black screen for 100ms. Then, a target appeared until the participant gave a response or for 2000ms. Hence, stimulus onset asynchrony (SOA) was 300ms. The inter-trial interval was 2000ms. Participants were informed about the end of each block and were given the opportunity to take a brief break before starting the next block of trials. They could begin with the next block independently by pushing the spacebar. The first trial of each block began after 4000ms, so participants had enough time to put their fingers on the buttons again.

After APT-1, the first explicit testing phase was conducted. Participants were instructed to rate the movements shown in the pictures (2 CS-neg/pos; 2 CS-pos/neg) according to their first and spontaneous impression of valence, harmfulness, and pain on VASs from 0 to 100, corresponding with the *stimulus selection phase*. Participants made 12 total ratings. The presentation order of the stimuli was randomized, while the rating scales appeared always in the same order: (1) valence, (2) harmfulness, (3) pain.

After the first testing phase, participants began the CC phase. The experimental parameters in the CC phase were the same as in the EC phase except that the CS-US contingency was reversed. During the EC phase, CS-neg/pos were paired with negative US and CS-pos/neg with positive US, whereas during the *CC phase* the valence of the US was switched. All US were new to the participants and were not used in any previous part of the study.

Next, participants performed the second implicit testing phase (APT-2). The experimental parameters and instructions were identical to APT-1. Again, participants began with a practice trial in which they were given feedback. If at least 80% of the answers were correct, they began with the actual APT. To avoid learning effects that could skew reaction times, words from the second word list were used as targets.

After completing the APT-2, participants were asked to rate the movements shown in the pictures (2 CS-neg/pos; 2 CS-pos/neg) a third time on the dimensions of valence, harmfulness, and pain. The presentation order of the pictures was randomized; otherwise this testing phase was identical to the one before.

After the experimental phase, participants were asked about their experiences with chronic back pain and whether they had been treated for strong back pain.

After the experiment, the experimenter explained the aims and the background of the study. Participants were given information about fear-avoidance beliefs, the fear-avoidance model, and recent scientific knowledge about back-stressing movements and chronic back pain.

Results – Study 1

Data reduction and analysis

Data from the APTs were analyzed using a 2x2x2 repeated-measures ANOVA with trial type (congruent, incongruent), target valence (positive, negative), and time point (EC phase, CC phase) as within-participants factors. The results were further examined using simple main effect analyses.

Data from explicit ratings were analyzed using a 3x2x3 repeated-measures ANOVA with dimension (valence, harmfulness, pain), CS type (CS-neg/pos; CS-pos/neg), and time point (baseline, EC phase, CC phase) as within- participants factors. The results were further examined using simple main effect analyses.

Bonferroni corrections were used for all analyses.

As reported in previous affective priming experiments [21,22], reaction times shorter than 250ms and larger than 1500ms were excluded from analyses to reduce the influence of outlier responses (APT-1: 0.1%; APT-2: 0.25%; total: 0.15%). Furthermore, reaction times from incorrect responses (e.g., response 'negative' if target valence was positive; APT-1: 2.04%; APT-2: 2.53%; total: 2.28%) and omissions (no response in time; APT-1: 0.06%; APT-2: 0.11%; total: 0.08%) were excluded from analyses. 2.55% of the data was excluded in total, which is comparable to other affective priming studies (e.g., [14]: 2.01%; [56]: 2.68%).

Implicit testing

For the APT data, the ANOVA revealed a significant three-way-interaction for trial type x target valence x time point, $F(1,32) = 7.58$, $p < .05$. Simple main effect analyses showed a significant difference only between trial types for negative targets in the APT-1: reaction times for negative targets in the APT-1 were shorter for congruent trials compared to incongruent trials, $F(1,32) = 5.74$, $p < .05$. No other significant main effect was found. The results are displayed in Figure 2 and Figure 3.

[Insert Figure 2 and Figure 3 around here]

Explicit testing

Analyses revealed a significant CS type X time point interaction, $F(2,64) = 4.51$, $p < .05$. Furthermore, results showed a significant main effect for dimension, $F(2,64) = 4.64$, $p < .05$. Based on these results, repeated-measures ANOVAs for all dimensions were conducted separately as post-hoc analyses.

Explicit testing – CS ratings for valence.

A repeated-measures ANOVA for the CS ratings for valence revealed a significant main effect for time point, $F(2,54) = 6.43$, $p < .01$, indicating more negative ratings of the movements over the course of the study (mean at baseline = 52; EC phase = 59; CC phase = 60), and a marginally significant CS-type x time point interaction, $F(2,64) = 3.00$, $p = .057$. Simple main effect analyses indicated that the predicted difference between CS-neg/pos and CS-pos/neg was significant only after the EC phase, $F(1,32) = 6.35$, $p < .05$, but not at baseline ($F(1,32) = 0.54$, $p = .468$), or after the CC phase ($F(1,32) = 0.00$, $p = .965$). These results indicate that CS-neg/pos were rated as more negative than CS-pos/neg

after acquisition (EC phase) only. There were no differences in ratings at baseline or after the CC phase.

CS-neg/pos ratings became more negative from baseline to the EC phase, $F(2,31) = 7.05, p < .01$, but no change in valence occurred from the EC phase to the CC phase. Ratings on valence for CS-pos/neg did not change from baseline to the EC phase, but were rated as more negative from the EC phase to the CC phase, $F(2,31) = 4.19, p < .05$. Thus, the marginally significant interaction can be attributed to changes in the negative direction, but not in the positive direction. The results for valence are displayed in Figure 4.

The main effect for CS type was not significant, $F(1,32) = 2.74, p = .11$.

Participants' attitudes seem to have been conditioned in the negative direction, but not in the positive direction, as indicated by changes in CS-neg/pos from baseline to acquisition as well as in changes in CS-pos/neg from acquisition to counter conditioning. There was no significant change in a positive direction for any of the CS.

[Insert Figure 4 around here]

Explicit testing – CS ratings for harmfulness.

A repeated-measures ANOVA for CS ratings for harmfulness revealed a significant main effect only for time point, $F(1,42) = 17.20, p < .01$, indicating a trend to rate all CS as more harmful over the course of the study. The main effect for CS type, $F(1,32) = 3.04, p = .09$, and the time point x CS type interaction, $F(2,60) = 2.17, p = .13$, were not significant. Thus, there were no differences between CS-neg/pos and CS-pos/neg at any time point in the course of the study. On a descriptive level, CS-neg/pos were rated as more harmful from baseline to the EC phase, but no changes occurred from the EC phase to the CC phase. No CS-pos/neg stimuli were rated as less harmful after the EC phase and no CS-neg/pos were rated as less harmful after the CC phase, indicating that conditioning of attitudes in a positive direction did not occur. The results for harmfulness are displayed in Figure 5.

[Insert Figure 5 around here]

Explicit testing – CS ratings for pain.

A repeated-measures ANOVA for CS ratings for pain revealed a significant main effect for time point, $F(1,32) = 9.49, p < .01$, indicating a trend to rate all CS as more painful over the course of the study. Furthermore, the time point x CS-type interaction was significant, $F(2,59) = 5.94, p < .01$. Simple main effect analyses showed no significant differences between CS-neg/pos and CS-pos/neg at baseline, $F(1,32) = 1.19, p = .284$, or after the CC phase, $F(1,32) = 0.56, p = .458$, but a significant difference in

CS ratings was found after the EC phase, $F(1,32) = 10.50$, $p < .01$, with CS-neg/pos rated as more painful than CS-pos/neg.

CS-neg/pos were rated as more painful from baseline to the EC phase, $p < .01$, but no change occurred from the EC phase to the CC phase. Ratings on pain for CS-pos/neg did not change from baseline to the EC phase, but CS-pos/neg were rated as more painful from the EC phase to the CC phase, $p < .01$. These results indicate rating changes only in a negative direction (i.e., more pain) for the movements depicted. The results for pain are displayed in Figure 6.

The main effect for CS type was not significant, $F(1,32) = 3.61$, $p = .07$.

[Insert Figure 6 around here]

Discussion – Study 1

Summary of aims and hypotheses. Study 1 tested whether evaluative conditioning affected attitudes towards back-stressing movements to examine learning mechanisms of fear-avoidance beliefs. First, it was expected that CS-neg/pos would be evaluated as more negative, more harmful, and more painful after the EC phase due to being paired with negatively valenced US; therefore, we expected that CS-neg/pos could be used as negative primes in APT-1 (vice versa for CS-pos/neg). A perfect affective priming effect, i.e. a main effect for congruency, would confirm a successful EC procedure. Second, it was hypothesized that the CC procedure would produce extinction learning, such that the CS-neg/pos and CS-pos/neg would be evaluated neutrally – that is, similar to baseline. This would result in the absence of an affective priming effect after the CC procedure. For explicit attitudes, we expected no differences between CS-neg/pos and CS-pos/neg on any of the dimensions.

Explicit EC effects for valence, harmfulness, and pain. There was a significant difference between CS-neg/pos and CS-pos/neg for ratings of painfulness of a movement. As expected, CS-neg/pos were rated as more painful than CS-pos/neg after the EC phase only. For ratings of valence, a trend was found but the difference did not reach significance. This might be due to limited power resulting from small sample size. No differences were found for harmfulness. It is noteworthy that the effects for pain (and valence) were due to the fact that CS-neg/pos were rated as more negative (more painful), but not because CS-pos/neg were rated as more positive (less painful) compared to baseline.

Implicit EC effects. Contrary to hypotheses, we did not find a perfect affective priming effect. Overall, reaction times for congruent trials were not significantly shorter than for incongruent trials after the evaluative conditioning procedure. The hypothesized difference between congruent and incongruent trials was found only for negative target valences. The evaluation of negative targets was found to be

slower than the evaluation of positive targets. This is consistent with results of other affective priming studies [14,43,47].

Explicit and implicit CC effects. After the counter conditioning procedure, there were no differences between CS-neg/pos and CS-pos/neg, either implicitly or explicitly.

Conclusion. Both the explicit and implicit data support the idea that the EC procedure seemed to produce attitude shifts only in the negative direction. Based on the theoretical background, we assume that none of the CS used as primes in APT-2 were affectively different from one another; this is supported by the fact that, after the CC phase, no differences were found on explicit attitudes between CS-neg/pos and CS-pos/neg. Again, these results only emerged due to “negative acquisition.” There were no indications of positive attitude formation. These results suggest that Evaluative Conditioning might lead to a negative shift in attitudes towards back-stressing movements as found in FAB.

Due to the partially unexpected results, we sought to replicate the findings using a larger sample. Furthermore, we aimed to assess the adequacy of the US used in the study by asking participants to rate the US on valence and arousal.

Study 2

Methods

Participants

The sample consisted of 50 students (76% female /38 female) from Philipps-University Marburg, who participated in the study in exchange for course credit points. The mean age was 23.24 years ($SD = 4.53$; range: 18-47). 52% of the participants were studying Psychology as a major. All participants gave informed consent and none were aware of the research hypotheses. This study was approved by the Ethics Committee of the Faculty of Psychology of the Philipps-University Marburg.

Procedure

Materials and procedures were almost identical to the first study. One US was replaced in the second study³ to increase the valence and arousal ratings of the positive US set. All other US, CS, and target words as well as experimental parameters (e.g., duration of trials) and phases were identical to those of the first study. US ratings for the dimensions of valence and arousal were conducted at the end of the second testing phase in order to keep the course of the study as similar as possible.

³ Picture number 8540 (old) was exchanged with picture number 8200 (new) from the IAPS.

To ensure the accuracy of US valence and arousal scores for the IAPS pictures, participants were asked to rate all US for valence and arousal on a VAS ranging from 1 (= very negative; low arousal) to 9 (= very positive; high arousal), in accordance with to the original scorings by the developers of the IAPS.

Results – Study 2

Data reduction and analysis

As in prior affective priming studies as well as in study 1, reaction times lower than 250ms and above 1500ms were excluded from data analysis (APT-1: 0.19%; APT-2: 0.13%; total: 0.16%). Furthermore, data including incorrect responses (APT-1: 0.65%; APT-2: 0.71%; total: 0.68%) and omissions (APT-1: 0%; APT-2: 0.02%; total: 0.01%) were excluded from analyses. In total, 0.84% of the data was excluded.

Data from APTs were analyzed using a 2x2x2 repeated-measures ANOVA with trial type (congruent, incongruent), target valence (positive, negative) and time point (EC phase, CC phase) as within-participants factors.

Data analyses for explicit ratings were performed as in study 1. Prior to analyzing every dimension separately, a 3x2x3 repeated-measures ANOVA was conducted. Next, the explicit CS ratings for valence, harmfulness, and pain were analyzed separately using a 3x2 repeated-measures ANOVA for each dimension, with time point (baseline, EC phase, CC phase) and CS type (CS-neg/pos, CS-pos/neg) as within-participants factors. The results were further examined using simple main effect analyses.

Bonferroni corrections were used for all analyses.

Implicit testing

The 2x2x2 repeated-measures ANOVA revealed a significant main effect for target valence only, $F(1,49) = 11.44$, $p < .01$, indicating that participants responded faster when presented with positive targets than negative targets. No other significant main effects or interaction effects were found. These results are displayed in Figure 7 and Figure 8.

[Insert Figure 7 and Figure 8 around here]

Explicit testing

Analyses revealed a significant CS type X time point interaction, $F(2,98) = 4.18, p < .05$. There was a significant main effect for dimension, $F(2,98) = 20.28, p < .001$. Based on these results, separate repeated-measures ANOVAs for each dimension were conducted as post-hoc analyses.

Explicit testing – CS ratings for valence.

A repeated-measures ANOVA for the CS ratings for valence revealed a significant main effect for time point, $F(2,98) = 20.23, p < .01$, indicating more negative ratings of the movements over the course of the study (mean at baseline = 46; EC phase = 56; CC phase = 57), and a significant CS type x time point interaction, $F(2,64) = 6.10, p < .01$. Simple main effect analyses revealed the expected significant difference between CS-neg/pos and CS-pos/neg only after the EC phase, $F(1,48) = 11.70, p < .05$, but not at baseline or after the CC phase. These results indicate that CS-neg/pos were rated more as negative than CS-pos/neg after acquisition (EC phase) only. There were no differences in ratings at baseline or after the CC phase.

CS-neg/pos were rated as more negative from baseline to the EC phase, $p < .01$, but no change occurred from the EC phase to the CC phase. Ratings of valence for CS-pos/neg did not change from baseline to the EC phase, but became more negative from the EC phase to the CC phase, although this difference did not reach significance, $p = .07$. The results indicate rating changes only in the negative direction, but no changes in the positive direction. The results for valence are displayed in Figure 9.

The main effect for CS type was not significant, $F(1,32) = 3.37, p = .07$.

Participants seem to have been conditioned negatively but not positively, as indicated by the changes in CS-neg/pos from baseline to the EC phase. There was no significant change in a positive direction for any of the CS.

[Insert Figure 9 around here]

Explicit testing – CS ratings for harmfulness.

A repeated-measures ANOVA for CS ratings for harmfulness revealed a significant main effect only for time point, $F(2,98) = 19.71, p < .01$, indicating a tendency to rate all CS as more harmful over the course of the study. The main effect for CS type, $F(1,49) = 0.53, p = .47$, and the time point x CS-type interaction, $F(2,98) = 1.92, p = .16$, were not significant. Thus, there were no differences between CS-neg/pos and CS-pos/neg at any study time point. On a descriptive level, CS-neg/pos showed a greater increase in harmfulness ratings from baseline to the EC phase compared to CS-pos/neg, but this

change was not significant. Participants rated all CS as more harmful after the EC phase and CC phase. No CS-pos/neg were rated as less harmful after the EC phase and no CS-neg/pos were rated as less harmful after the CC phase, indicating that participants' attitudes were not conditioned positively. The results for harmfulness are displayed in Figure 10.

[Insert Figure 10 around here]

CS ratings for pain.

A repeated-measures ANOVA for CS ratings for pain revealed a significant main effect for time point, $F(2,98) = 12.18, p < .01$, indicating a tendency to rate all CS as more painful over the course of the study. Furthermore, the time point x CS type interaction was significant, $F(2,59) = 3.48, p < .05$. Simple main effect analyses showed no significant differences between CS-neg/pos and CS-pos/neg at baseline, $p = .809$, or after the CC phase, $p = .883$, but a significant difference was found for CS ratings after the EC phase, $p < .01$, with CS-neg/pos rated as more painful than CS-pos/neg.

CS-neg/pos were rated more as painful from baseline to the EC phase, $p < .01$, but no change occurred from the EC phase to the CC phase. Ratings of pain for CS-pos/neg did not change from baseline to the EC phase, but increased from the EC phase to the CC phase, $p < .05$. These results indicate rating changes only in the negative direction (i.e., more pain) for the movements, but no changes in the positive direction. The results for pain are displayed in Figure 11.

The main effect for CS type was not significant, $F(1,49) = 2.65, p = .11$.

[Insert Figure 11 around here]

US ratings for valence and arousal

Participants rated US with an expected positive valence as significantly more positive ($M = 7.61, SD = 1.08$) than US with an expected negative valence ($M = 1.83, SD = 0.67$), $t = -28.32, p < .01$. The affective extremity for US valence was higher for negatively valenced US ($M = 3.18, SD = 0.68$) than for positively valenced US ($M = 2.61, SD = 1.08$), $t = 3.73, p < .01$.

Both US types were rated as moderately arousing (positive US: $M = 5.30, SD = 1.78$; negative US: $M = 5.81, SD = 1.84$), and this difference was not significant. Affective extremity for arousal ratings also did not differ across US types, $t = -1.40, p = .169$.

These results suggest that the US selected were appropriate and that participants reliably differentiated between US with positive and negative valences, whereas ratings for arousal did not differ significantly between positively and negatively valenced US.

Study 2 – Discussion

Summary of aims. The goal of Study 2 was to replicate the findings from Study 1, in which data on implicit and explicit attitudes toward back-stressing movements indicated that evaluative conditioning produced attitude shifts only in the negative direction. A similar pattern of conditioning in the negative direction was found for the counter conditioning procedure.

Explicit EC effects for valence, harmfulness and pain. Results of Study 2 indicated EC effects on explicit attitudes in the negative direction, but not the positive direction, on the dimensions of valence and pain; i.e., CS-neg/pos were rated as more negative and more painful after the EC phase, whereas CS-pos/neg were rated similarly to baseline. No significant effects were found for harmfulness, although descriptive data suggested a pattern similar to that found for valence and pain. The effects for pain and valence appear to be accounted for by negative shifts on ratings of CS-neg/pos rather than by positive shifts on ratings of CS-pos/neg compared to baseline.

Implicit EC effects. Contrary to our hypotheses and results in Study 1, the affective priming data indicated that reaction times in congruent trials were not shorter than in incongruent trials, and no interaction was found. This is of particular interest given that the explicit attitudes data would suggest that CS-neg/pos would be appropriate negative primes; thus, we would expect participants to respond faster to negative targets preceded by these primes on the affective priming task. Nevertheless, the only significant result across both affective priming tasks was the difference in reaction times between positive and negative targets. While contrary to our hypotheses, this result is consistent with previous literature [47,49].

Explicit and implicit CC effects. After the counter conditioning procedure, no differences between CS-neg/pos and CS-pos/neg were found on any of the dimensions measured to assess explicit attitudes. Again, this (non-)effect occurred because participants' ratings shifted in a negative direction; no learning in the positive direction was found. The implicit testing results in APT-2, after the CC phase, replicated the main effect for target valence from APT-1, indicating shorter reaction times for positive targets.

Adequacy of US. Participants' ratings of valence and arousal for US revealed that positive and negative US were perceived as equally affectively arousing, but were perceived as different with respect to affective extremity. Both results were unexpected: based on the norms reported in the IAPS manual, we expected higher levels of arousal for negative US, but similar levels of affective extremity. If negative US had been perceived as more strongly arousing than positive US, this might have been one explanation for why attitude shifts occurred only in a negative direction, as one might expect stronger reactions towards stronger US. However, results from US ratings do not support this

explanation, although it should be noted that these results are based on self-report measures and might not reflect actual physiological arousal.

Conclusion. The results of Study 2 suggest that evaluative conditioning may lead to a negative shift in explicit attitudes towards back-stressing movements. Our results do not indicate any learning on an implicit level or any conditioning of attitudes in a positive direction.

Sub-analyses and comparison of Studies 1 and 2.

Participants in both studies were healthy college students. We compared the samples on demographic characteristics, pain-related questionnaire scores, depression, and experiences with chronic low back pain through family members or friends. The study samples were comparable on all of the above characteristics except depression and experiences with CLBP, with the Study 1 sample showing higher levels of depression ($t = 1.37, p < .05$) and more experiences with CLBP ($Chi^2 = 7.18, p < .01$). A summary of similarities and differences across studies is provided in table 1.

[Insert Table 1 around here]

General discussion

We conducted two studies to examine whether an evaluative conditioning paradigm affects implicit and explicit attitudes towards back-stressing movements.

After acquisition, i.e. the EC phase, participants reported more negative attitudes towards back-stressing movements on self-report measures, and implicit assessment also indicated a negative attitude shift. Contrary to our expectations, no attitude shift in the positive direction occurred. These results suggest that FAB can develop without the induction of fear or direct experience of pain, whereas it appears more difficult to learn to view back-stressing movements positively.

Both studies indicated that the EC paradigm led to more negative explicit evaluations of the previously neutrally-rated movements, e.g., bending forward was evaluated as more negative and more painful after its pairing with negatively valenced US. When the same movements were paired with positively valenced US, a positive attitude shift was not found on any of the dimensions assessed at any time point in the experiments, including the acquisition phase in which positive US were used in the EC paradigm as well as the CC phase in which the CS-US contingencies were reversed.

In the first, although not in the second experiment, the data from implicit assessment of attitudes supports similar conclusions. A congruency effect for negative prime-target pairings was found after acquisition via the EC paradigm and declined after the counter conditioning. Thus, it appears that for

the first affective priming task, CS-neg/pos served as negative primes, whereas CS-pos/neg did not serve as positive primes, and therefore the only facilitation effect for prime-target congruency occurred for negative targets. No affective priming effect was found after counter conditioning, which is also in line with the explicit effects, since CS-neg/pos and CS-pos/neg no longer differed from one another. Instead, a significant main effect for target valence was found, indicating that participants evaluated positive targets more quickly than negative targets independently of the preceding prime. This result makes sense given the lack of a congruency effect; thus, no inhibition or facilitation effects for (in-)congruent pairs influenced response latencies. Valence effects showing faster reaction time for positive targets have also been found in other studies using affective priming tasks [38,47].

In sum, both studies found a negative shift in explicit attitudes towards back-stressing movements after an evaluative conditioning procedure, and Study 1 also found a negative shift in attitudes measured at the implicit level.

Possible explanations for the lack of positive conditioning may include the selection of US as well as a frequently documented negativity bias such that negative information, relative to positive information, is usually learned or detected faster and has stronger impact on behavior [4,45]. With respect to US selection, negative US were closely related to the topic of pain, e.g., a man lying in hospital bed, whereas positive US were chosen on the basis of matching arousal and valence ratings and may have been less likely to evoke associations with pain. Therefore, it is possible that it was easier to form associations between negatively valenced, pain-related US and CS showing back-stressing movements due to the similar content [10]. However, given that positive US were less adequate, this raises the question of whether the CC phase was instead an extinction phase in the classical sense, meaning the absence of an aversive US.

In a classical conditioning paradigm assuming an underlying expectancy-learning mechanism, the absence of an aversive US would be expected to result in extinction due to the development of a new CS-(no)US association [5,6], and thus, a neutral evaluation of the CS as previously found at baseline. The stable negative ratings found in our study are more in line with research on evaluative conditioning, which describes a resistance for EC effects to extinction in terms of unreinforced CS presentations [2,11]. Our results support the idea that a different learning mechanism underlies evaluative conditioning, i.e., referential learning rather than expectancy learning [20]. Expectancy learning refers to a learning mechanism in which the US (e.g., pain) is expected in the presence of the CS (e.g., movement) after the conditioning procedure, whereas in referential learning the association with the US is inferred in the presence of the CS, but “without [...] additionally generating the active expectancy of real US occurrence in the here-and-now of the immediate future” [15, p. 218].

Hermans and colleagues (2002) [20] compared expectancy learning and referential learning and found that these forms of classical conditioning can co-occur, but may have different implications with respect to interventions, particularly with regard to extinction processes. Outcomes of conditioning may also differ, e.g. higher fear responses were found for expectancy learning.

Fear of movement has been seen as the central component of the fear-avoidance model, which aims to explain the vicious cycle among avoidance, physical deconditioning, and pain [41]. Thus, interventions such as graded activity or exposure therapy have seemed promising for the treatment of CLBP, as these interventions involve approaching the fear-eliciting movements with the aim of reducing fear and avoidance. However, the role of fear in the development and maintenance of CLBP has been debated in the literature [41]. In an fMRI study, Barke and colleagues (2012) [3] did not find any group differences on neural correlates of pain responses among highly fearful/avoidant participants compared to participants low on measures of fear-avoidance or compared to healthy participants. A review by Pincus and colleagues (2002) [40] about fear of pain in patients with LBP questioned the predictive utility of fear with respect to back pain prognosis [42]. Instead, they postulate that health beliefs or depression might be more important in the transition from acute to chronic pain. In the experiments presented here, evaluative conditioning changed explicit beliefs or attitudes about back-stressing movements among healthy participants without the induction of fear. At the level of implicit attitudes, the attitude change occurred only in the first study. A closer look at the sample characteristics indicated that participants in the first study had higher scores for depression and were more experienced with CLBP among family members or friends. Considering the proposed pathways for the transition from acute to chronic pain by Pincus and colleagues (2002) [40], it could be argued that the implicit effects in the negative direction found in Study 1 may be attributable to more depression or negative affect or to stronger preconceptions about back-stressing movements due to more experience with CLBP. However, differences between implicit and explicit results might also be due to the manner in which we assessed EC effects. In general, EC effects have found to be smaller when assessed implicitly, and effects on the APT have been shown to be smaller compared to other implicit assessment methods. Thus, it is possible that the explicit ratings are more reliable.

Clinical and scientific implications.

The current results have two important implications for clinicians. First, clinicians should be aware of negative beliefs in patients with acute or chronic low back pain. Our results indicate that even brief experiences with negative associations with back-stressing movements – which may happen without awareness – might lead to effects on attitudes that could result in avoidance and disability over the long term [41,42]. Second, the lack of extinction in both studies suggests that changing negative

beliefs about back-stressing movements is more difficult than acquiring these beliefs. This speaks to the importance of preventing the development of FAB, perhaps by proactive psychoeducation and stronger consolidation of experiences that disconfirm FAB. Furthermore, although exposure therapies and graded activity show promising effects in the treatment of CLBP, the present results suggest that the focus of those interventions should move from mere fear reduction to corrective experiences (expectancy violation). This is consistent with the approach to exposure recommended by Craske and colleagues (2008), in which inhibitory learning is emphasized [8]. Our results support the idea that fear is not necessary for the development of attitudes towards back-stressing movements, and thus, fear might not be the most important target in the treatment of CLBP.

Further research is needed to better understand the relevance of FAB or other cognitive components to chronic pain. The results of the two studies presented here underscore the importance of the call for replication studies in psychology to strengthen experimental findings [1]. The differences between the two studies with respect to implicit attitude changes highlight the fact that similar procedures among apparently similar samples may produce divergent results, perhaps due to unexpected or unrecognized sample differences. Further investigation of FAB and other cognitive aspects of chronic pain should involve assessment of both implicit and explicit attitudes. In addition, an innovative aspect of the current research is the finding that attitudes toward movements can be formed on dimensions other than simply valence, including assessments of expected pain and harmfulness, and this type of attitude formation should be further investigated in research on chronic pain.

Limitations.

These studies have several limitations. First, as previously mentioned, there is no baseline data for implicit measures. Thus, given the differences between the implicit and explicit results, we cannot be sure that participants' baseline implicit ratings were neutral, as measured explicitly. Further studies could address this problem by including an APT at baseline. This baseline data would help to place results after the EC procedure in context. Second, US in the first experiment were chosen using the norms presented in the IAPS manual [32]. Results in the second experiment indicated that the normative data did not fully match the current study sample's reaction to the stimuli. Thus, valence and arousal ratings for stimuli should be assessed in every experiment, ideally. Furthermore, to ensure the similarity of positive and negative US, both sets of US should depict content related to the topic of pain; positive US might include stimuli such as pictures of massages or physiotherapeutic interventions. Arousal could be further assessed using psychophysiological measures, e.g. electrodermal activity or heart rate variability. Third, because the CS used here were developed for this study, additional validation research is needed. It would also be of interest to ask participants

what they rated when they evaluated the stimuli: although they were asked to rate the movement shown in the picture, it is possible that participants rated the picture globally.

Conclusion.

To our knowledge, this is the first study to examine evaluative conditioning as a learning mechanism for fear-avoidance beliefs measured implicitly and explicitly. Results indicated that EC influenced on attitude formation towards back-stressing movements even without the induction of pain or fear. This may help to explain why pain-free people endorse fear-avoidance beliefs without the direct experience of a back-related trauma. Furthermore, results indicated a negativity bias, which may be of particular interest in the further study of chronic pain.

- [1] Asendorpf JB, Conner M, de Fruyt F, de Houwer J, Denissen J, Fiedler K, Fiedler S, Funder DC, Kliegl R, Nosek BA, Perugini M, Robert BW, Schmitt M, Vanaken MAG, Weber H, Wicherts JM. Recommendations for increasing replicability in psychology. *Eur. J. Pers.* 2012;119:108–119. doi:10.1002/per.
- [2] Baeyens F, Díaz E, Ruiz G. Resistance to extinction of human evaluative conditioning using a between-subjects design. *Cogn. Emot.* 2005;19:245–68. doi:10.1080/02699930441000300.
- [3] Barke A, Baudewig J, Schmidt-Samoa C, Dechent P, Kröner-Herwig B. Neural correlates of fear of movement in high and low fear-avoidant chronic low back pain patients: An event-related fMRI study. *Pain* 2012;153:540–552. doi:10.1016/j.pain.2011.11.012.
- [4] Baumeister RF, Bratslavsky E, Finkenauer C, Vohs KD. Bad Is Stronger Than Good. *Rev. Gen. Psychol.* 2001;5:323–370.
- [5] Bouton ME. A learning theory perspective on lapse, relapse, and the maintenance of behavior change. *Heal. Psychol.* 2000;19:57–63. doi:10.1037/0278-6133.19.1.
- [6] Bouton ME, Swartzentruber D. Sources of relapse after extinction in Pavlovian and instrumental learning. *Clin. Psychol. Rev.* 1991;11:123–140.
- [7] Buer N, Linton SJ. Fear-avoidance beliefs and catastrophizing: Occurrence and risk factor in back pain and ADL in the general population. *Pain* 2002;99:485–491.
- [8] Craske MG, Kircanski K, Zelikowsky M, Mystkowski J, Chowdhury N, Baker A. Optimizing inhibitory learning during exposure therapy. *Behav. Res. Ther.* 2008;46:5–27.
- [9] Crombez G, Eccleston C, Van Damme S, Vlaeyen JWS, Karoly P. Fear-avoidance model of chronic pain: the next generation. *Clin. J. Pain* 2012;28:475–83. doi:10.1097/AJP.0b013e3182385392.
- [10] Garcia J, Koelling RA. Relation of cue to consequence in avoidance learning. *Psychon. Soc.* 1966;4:123–124.
- [11] Gawronski B, Gast A, De Houwer J. Is evaluative conditioning really resistant to extinction? Evidence for changes in evaluative judgements without changes in evaluative representations. *Cogn. Emot.* 2014;9931:1–15. doi:10.1080/02699931.2014.947919.
- [12] Glombiewski JA, Riecke J, Holzapfel S, Rief W, König S, Lachnit H, Seifart U. Do chronic pain patients show autonomic arousal when confronted with feared movements? An experimental investigation of the fear-avoidance model. *Pain* 2015;156:547–554.
- [13] Goubert L, Crombez G, De Bourdeaudhuij I. Low back pain, disability and back pain myths in a community sample: Prevalence and interrelationships. *Eur. J. Pain* 2004;8:385–394.
- [14] Goubert L, Crombez G, Hermans D, Vanderstraeten G. Implicit attitude towards pictures of back-stressing activities in pain-free subjects and patients with low back pain: An affective priming study. *Eur. J. Pain* 2003;7:33–42.
- [15] Goubert L, Crombez G, Peters M. Pain-related fear and avoidance: A conditioning perspective. In: Asmundson GJG, Vlaeyen JWS, Crombez G, editors. *Understanding and Treating Fear of Pain*. New York: Oxford University Press, 2004. pp. 25–50.
- [16] Goubert L, Vlaeyen JWS, Crombez G, Craig KD. Learning about pain from others: An observational learning account. *J. Pain* 2011;12:167–174. doi:10.1016/j.jpain.2010.10.001.
- [17] Grotle M, Vøllestad NK, Veierød MB, Brox JI. Fear-avoidance beliefs and distress in relation to disability in acute and chronic low back pain. *Pain* 2004;112:343–352.
- [18] Hautzinger M, Bailer M. *Allgemeine Depressions Skala – ADS*. Weinheim: Beltz, 1993 p.
- [19] Helsen K, Goubert L, Peters ML, Vlaeyen JWS. Observational learning and pain-related fear: An experimental study with colored cold pressor tasks. *J. Pain* 2011;12:1230–1239. doi:10.1016/j.jpain.2011.07.002.
- [20] Hermans D, Crombez G, Vansteenwegen D, Baeyens F, Eelen P. Expectancy-learning and evaluative learning in human classical conditioning: Differential effects of extinction. *Adv. Psychol. Res.* Vol. 12 2002;40:17–40.
- [21] Hermans D, De Houwer J, Eelen P. A time course analysis of the affective priming effect. *Cogn. Emot.* 2001;15:143–165. doi:10.1080/0269993004200033.
- [22] Hermans D, Houwer J De, Eelen P. The affective priming effect: Automatic activation of

- evaluative information in memory. *Cogn. Emot.* 1994;8 VN-re:515–533. doi:10.1080/02699939408408957.
- [23] Hofmann W, De Houwer J, Perugini M, Baeyens F, Crombez G. Evaluative conditioning in humans: A meta-analysis. *Psychol. Bull.* 2010;136:390–421. doi:10.1037/a0018916.
 - [24] den Hollander M, de Jong JR, Volders S, Goossens ME, Smeets RJ, Vlaeyen JW. Fear reduction in patients with chronic pain: a learning theory perspective. *Expert Rev. Neurother.* 2010;10:1733–1745.
 - [25] den Hollander M, de Jong JR, Volders S, Goossens ME, Smeets RJ, Vlaeyen JW, Hollander MD, de Jong JR, Volders S, Goossens ME, Smeets RJ, Vlaeyen JW, den Hollander M, de Jong JR, Volders S, Goossens ME, Smeets RJ, Vlaeyen JW. Fear reduction in patients with chronic pain: a learning theory perspective. *Expert Rev. Neurother.* 2010;10:1733–1745. doi:10.1586/ern.10.115.
 - [26] Holzapfel S, Riecke J, Rief W, Schneider J, Glombiewski JA. Development and Validation of the Behavioral Avoidance Test – Back pain (BAT-Back) for Patients with Chronic Low Back Pain. *Clin. J. Pain* 2016;1. doi:10.1097/AJP.0000000000000349.
 - [27] Houwer J De, Thomas S, Baeyens F, De Houwer J, Thomas S, Baeyens F. Association learning of likes and dislikes: A review of 25 years of research on human evaluative conditioning. *Psychol. Bull.* 2001;127:853–869. doi:http://psycnet.apa.org/doi/10.1037/0033-2909.127.6.853.
 - [28] Ihlebaek C, Eriksen HR. Myths and perceptions of back pain in the Norwegian population, before and after the introduction of guidelines for acute back pain. *Scand. J. Public Health* 2005;33:401–406.
 - [29] Ito T a, Larsen JT, Smith NK, Cacioppo JT. Negative information weighs more heavily on the brain: the negativity bias in evaluative categorizations. *J. Pers. Soc. Psychol.* 1998;75:887–900.
 - [30] Jones CR, Olson MA, Fazio RH. Evaluative Conditioning: The “How” Question. *Adv. Exp. Soc. Psychol.* 2010;43:205–255.
 - [31] Kugler, K., Wijn, J., Geilen, M., de Jong, J., and Vlaeyen JWS. The Photograph series of Daily Activities (PHODA). 1999.
 - [32] Lang, P.J., Bradley, M.M., & Cuthbert BN. International affective picture system (IAPS): Affective ratings of pictures and instruction manual. Technical Report A-8. University of Florida, 2008 p. Available: ftp://dhcp-129-105-171-164.psych.northwestern.edu/OpenShare/ESPN/IAPS 1-16/IAPS 1-16/IAPSmanual.pdf.
 - [33] Leeuw M, Goossens MEJB, Linton SJ, Crombez G, Boersma K, Vlaeyen JWS. The Fear-Avoidance Model of Musculoskeletal Pain: Current State of Scientific Evidence. *J. Behav. Med.* 2007;30:77–94. doi:10.1007/s10865-006-9085-0.
 - [34] McCracken LM. “Attention” to pain in persons with chronic pain: a behavioural approach. *Behav. Ther.* 1997;28:271–284.
 - [35] Meulders A, Vansteenwegen, Debora; Vlaeyen JWS. The acquisition of fear of movement-related pain and associative learning: A novel pain-relevant human fear conditioning paradigm. *Pain* 2011;152:2460–2469.
 - [36] Meyer K, Sprotta H, Mannion AF. Cross-cultural adaptation, reliability, and validity of the German version of the Pain Catastrophizing Scale. *J. Psychosom. Res.* 2008;64:469–478.
 - [37] Miller R, Kori S, Todd D. The Tampa Scale. 1991.
 - [38] Padovan C, Versace R, Thomas-Antérion C, Laurent B. Evidence for a selective deficit in automatic activation of positive information in patients with Alzheimer’s disease in an affective priming paradigm. *Neuropsychologia* 2002;40:335–339.
 - [39] De Peuter S, Van Diest I, Vansteenwegen D, Van Den Bergh O, Vlaeyen JWS. Understanding fear of pain in chronic pain: Interoceptive fear conditioning as a novel approach. *Eur. J. Pain* 2011;15:889–894.
 - [40] Pincus T, Burton a K, Vogel S, Field AP. A systematic review of psychological factors as predictors of chronicity/disability in prospective cohorts of low back pain. *Spine (Phila Pa 1976)* 2002;27:E109-20. doi:10.1097/00007632-200203010-00017.
 - [41] Pincus T, Smeets RJEM, Simmonds MJ, Sullivan MJL. The fear avoidance model disentangled:

- improving the clinical utility of the fear avoidance model. *Clin. J. Pain* 2010;26:739–746.
- [42] Pincus T, Vogel S, Burton AK, Santos R, Field AP. Fear avoidance and prognosis in back pain: A systematic review and synthesis of current evidence. *Arthritis Rheum.* 2006;54:3999–4010.
- [43] Raes AK, De Raedt R. The Effect of Counterconditioning on Evaluative Responses and Harm Expectancy in a Fear Conditioning Paradigm. *Behav. Ther.* 2012;43:757–767. doi:10.1016/j.beth.2012.03.012.
- [44] Riecke J, Holzapfel S, Rief W, Lachnit H, Glombiewski J a. Cross-cultural adaption of the German Quebec back pain disability scale: An exposure-specific measurement for back pain patients. *J. Pain Res.* 2016;9:9–15.
- [45] Rozin P, Royzman EB. Negativity Bias, Negativity Dominance, and Contagion Paul. *Personal. Soc. Psychol. Rev.* 2001;5:296–320.
- [46] Rusu A, Kreddig N, Hallner D, Al. E. Fear of movement/(Re)injury in low back pain: confirmatory validation of a German version of the Tampa Scale for Kinesiophobia. *BMC Musculoskelet Disord.* 2014;15.
- [47] Sollberger B, Rebe R, Eckstein D. Musical Chords as Affective Priming Context in a Word-Evaluation Task. *Music Percept. An Interdiscip. J.* 2003;20:263–282.
- [48] Sullivan MJL, Bishop SR, Pivik J. The Pain Catastrophizing Scale: Development and validation. *Psychol. Assess.* 1995;7:524–532.
- [49] Vermeulen N, Luminet O, Corneille O. Alexithymia and the automatic processing of affective information: evidence from the affective priming paradigm. *Cogn. Emot.* 2006;20:64–91.
- [50] Vlaeyen J, Jong J, Geilen M, Heuts P, van Breukelen G. The Treatment of Fear of Movement/(Re)injury in Chronic Low Back Pain: Further Evidence on the Effectiveness of Exposure In Vivo. *Clin. J. Pain* 2002;18:251–261. doi:10.1097/00002508-200207000-00006.
- [51] Vlaeyen JWS, De Jong J, Geilen M, Heuts PHTG, Van Breukelen G. Graded exposure in vivo in the treatment of pain-related fear: A replicated single-case experimental design in four patients with chronic low back pain. *Behav. Res. Ther.* 2001;39:151–166.
- [52] Vlaeyen JWS, Linton SJ. Fear-avoidance and its consequences in chronic musculoskeletal pain: A state of the art. *Pain* 2000;85:317–332.
- [53] Vlaeyen JWS, Linton SJ. Fear-avoidance model of chronic musculoskeletal pain: 12 years on. *Pain* 2012;153:1144–1147. doi:10.1016/j.pain.2011.12.009.
- [54] Vö ML-H, Conrad M, Kuchinke L, Urton K, Hofmann MJ, Jacobs AM. The Berlin Affective Word List Reloaded (BAWL-R). *Behav. Res. Methods* 2009;41:534–538. doi:10.3758/BRM.41.2.534.
- [55] Waddell G, Newton M, Henderson I, Somerville D, Main CJ. A Fear-Avoidance Beliefs Questionnaire (FABQ) and the role of fear-avoidance beliefs in chronic low back pain and disability. *Pain* 1993;52:157–168.
- [56] Wentura D. Dissociative affective and associative priming effects in the lexical decision task: yes versus no responses to word targets reveal evaluative judgment tendencies. *J. Exp. Psychol. Learn. Mem. Cogn.* 2000;26:456–469.
- [57] Wertli MM, Rasmussen-Barr E, Weiser S, Bachmann LM, Brunner F. The role of fear avoidance beliefs as a prognostic factor for outcome in patients with nonspecific low back pain: a systematic review. *Spine J.* 2014;14:816–36.e4. doi:10.1016/j.spinee.2013.09.036.
- [58] Woby SR, Watson PJ, Roach NK, Urmston M. Are changes in fear-avoidance beliefs, catastrophizing, and appraisals of control, predictive of changes in chronic low back pain and disability? *Eur. J. Pain* 2004;8:201–210.

Figure 1 Procedure of the study.

Stimulus selection phase (online)	1 week	Experimental phase (laboratory)						
Baseline assessment		Evaluative Conditioning	First testing phase		Counter Conditioning	Second testing phase		Post assessment
<ul style="list-style-type: none">CS ratings at baselineQuestionnairesDemographics		CS-US pairings	Implicit testing: APT-1	Explicit testing: CS ratings	CS-US pairings with reversed contingencies	Implicit testing: APT-2	Explicit testing: CS ratings	<ul style="list-style-type: none">US ratingsExperiences with CLBP

Notes: CS = conditioned stimulus; US = unconditioned stimulus; APT = Affective Priming Task; CLBP = Chronic Low Back Pain

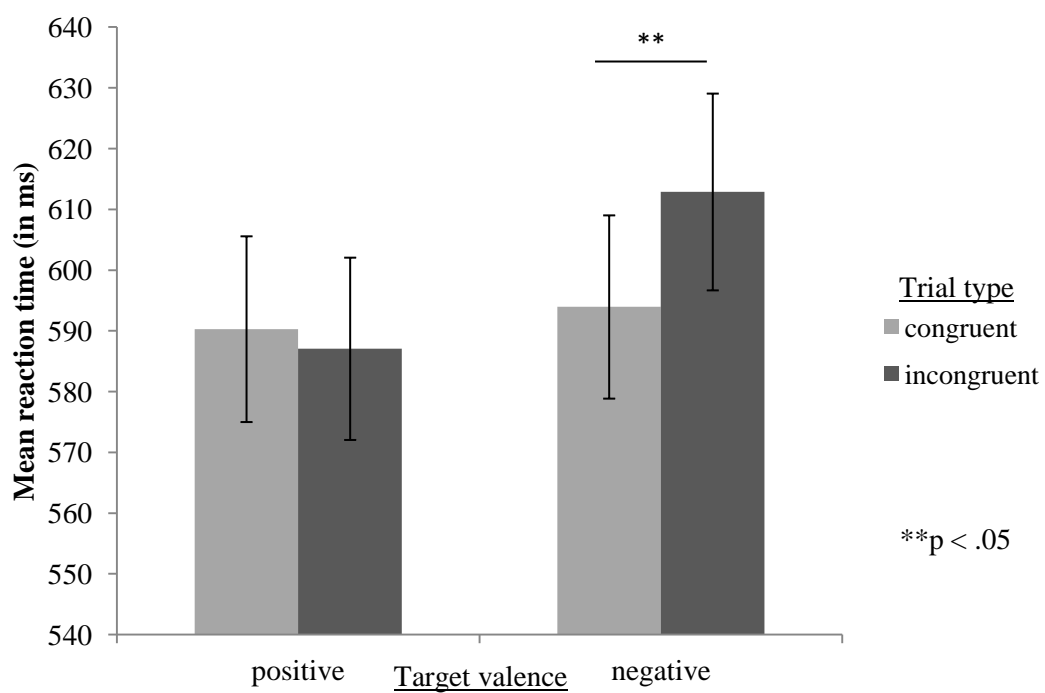


Figure 2: Mean reaction times for congruent (CS-neg/pos_negative target & CS-pos/neg_positive target) and incongruent (CS-neg/pos_positive target & CS-pos/neg_negative target) trials subdivided by target valence for APT-1.

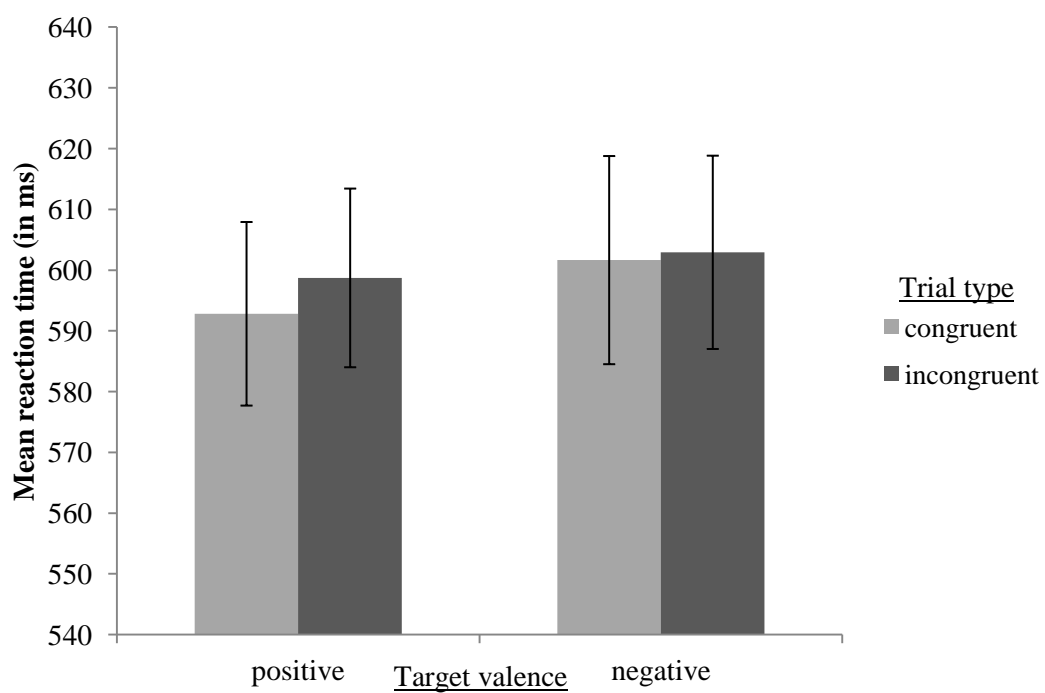


Figure 3: Mean reaction times for congruent (CS-pos/neg_negative target & CS-neg/pos_positive target) and incongruent (CS-pos/neg_positive target & CS-neg/pos_negative target) trials divided by target valence for APT-2.

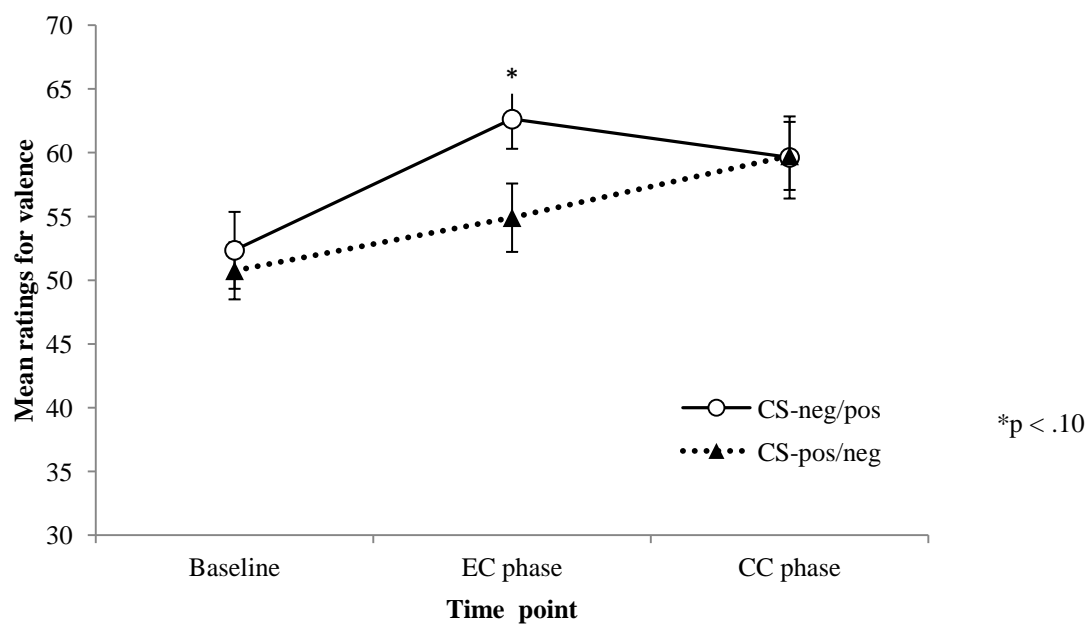


Figure 4: CS ratings for valence during the experiment (higher values indicating more negative ratings).

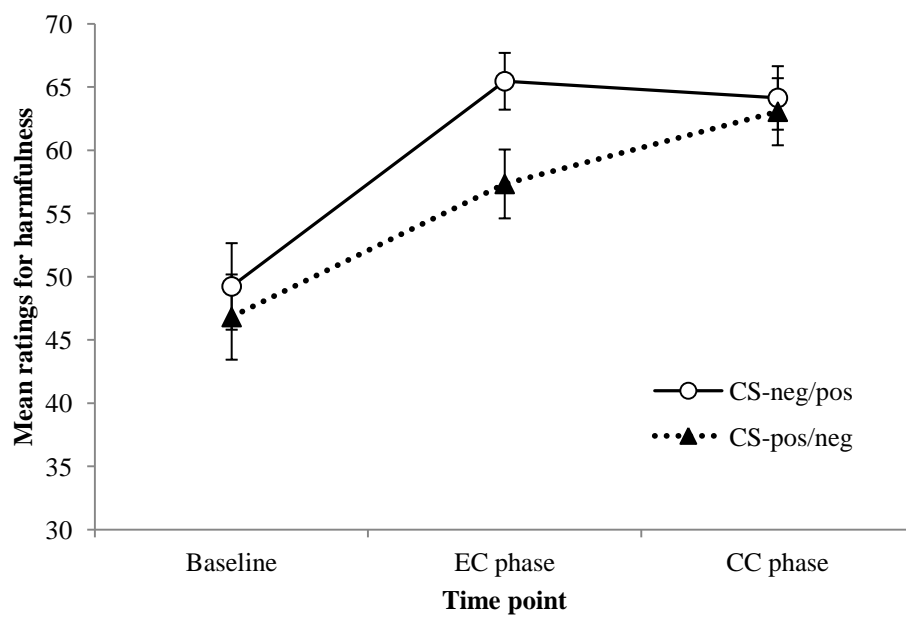


Figure 5: CS ratings for harmfulness during the experiment (higher values indicating more harmful ratings).

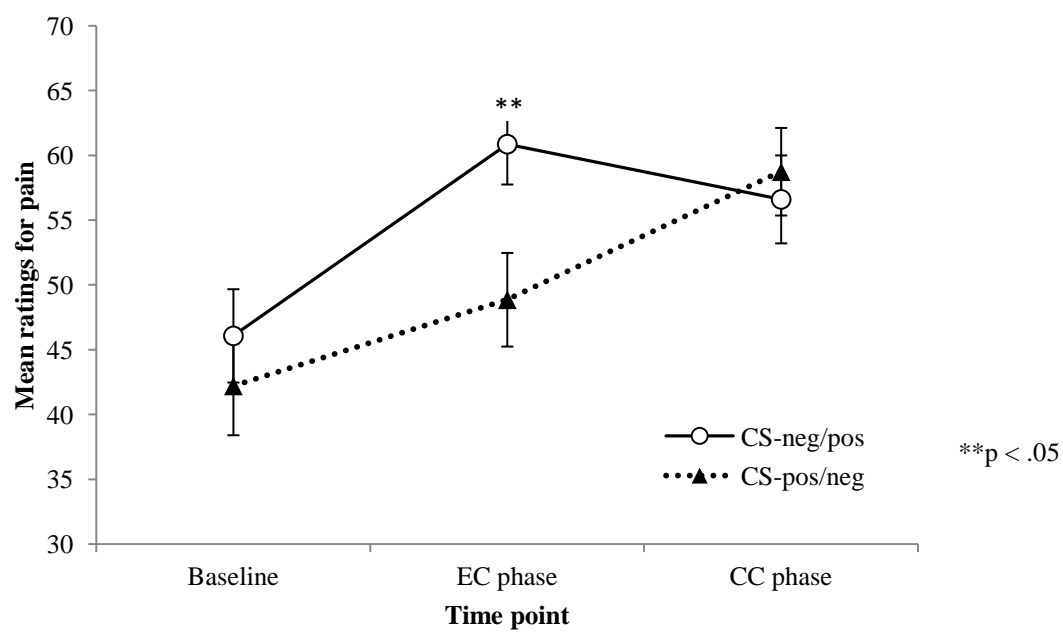


Figure 6: CS ratings for pain during the experiment (higher values indicating more painful ratings).

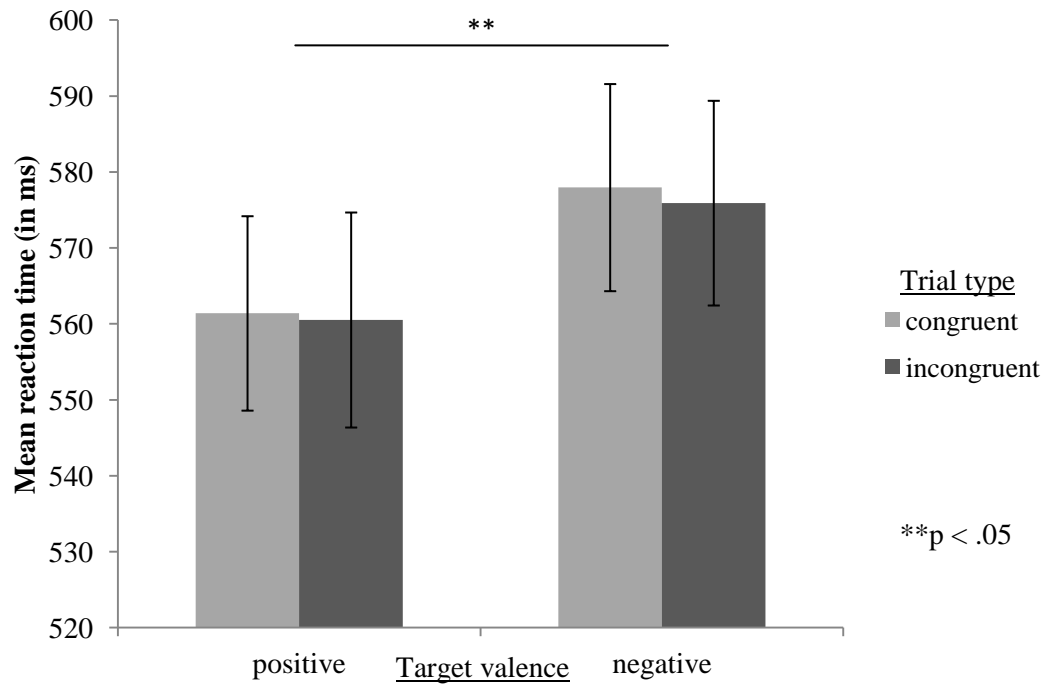


Figure 7: Mean reaction times for congruent (CS-neg/pos_negative target & CS-pos/neg_positive target) and incongruent (CS-neg/pos_positive target & CS-pos/neg_negative target) trials subdivided by target valence for APT-1.

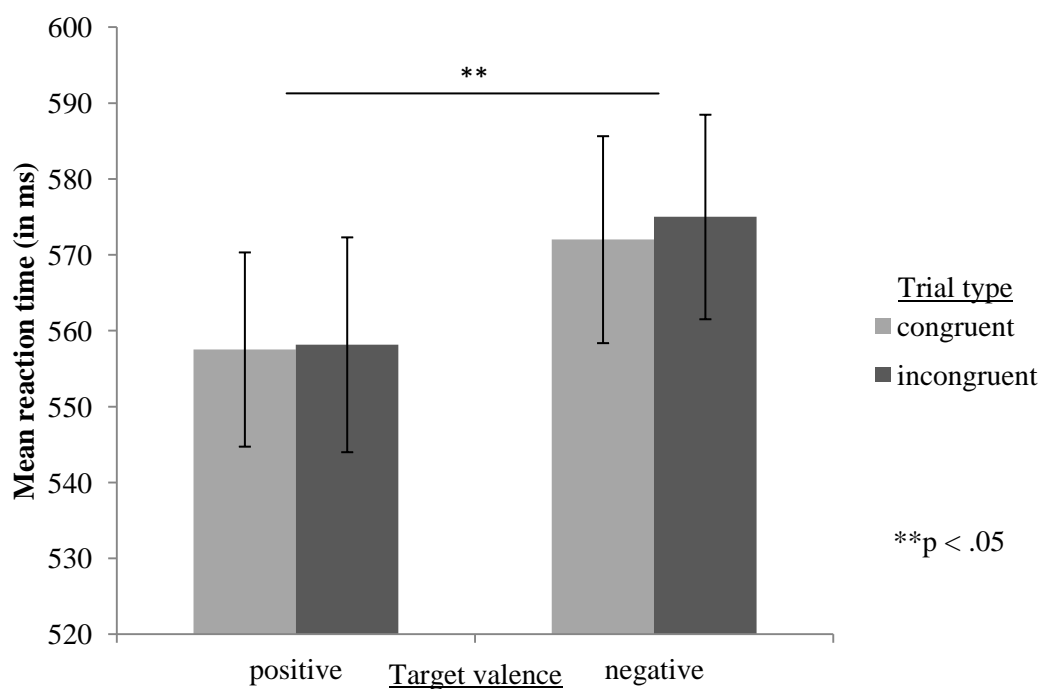


Figure 8: Mean reaction times for congruent (CS-pos/neg_negative target/CS-neg/pos_positive target) and incongruent (CS-pos/neg_positive target/CS-neg/pos_negative target) trials divided by target valence for APT-2.

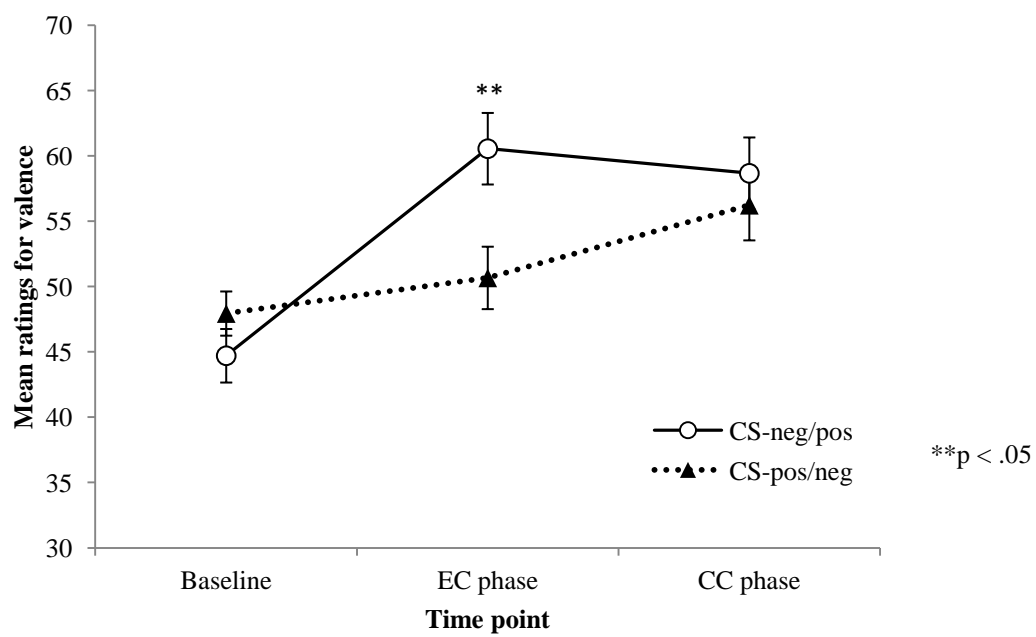


Figure 9: CS ratings for valence during the experiment (higher values indicating more negative ratings).

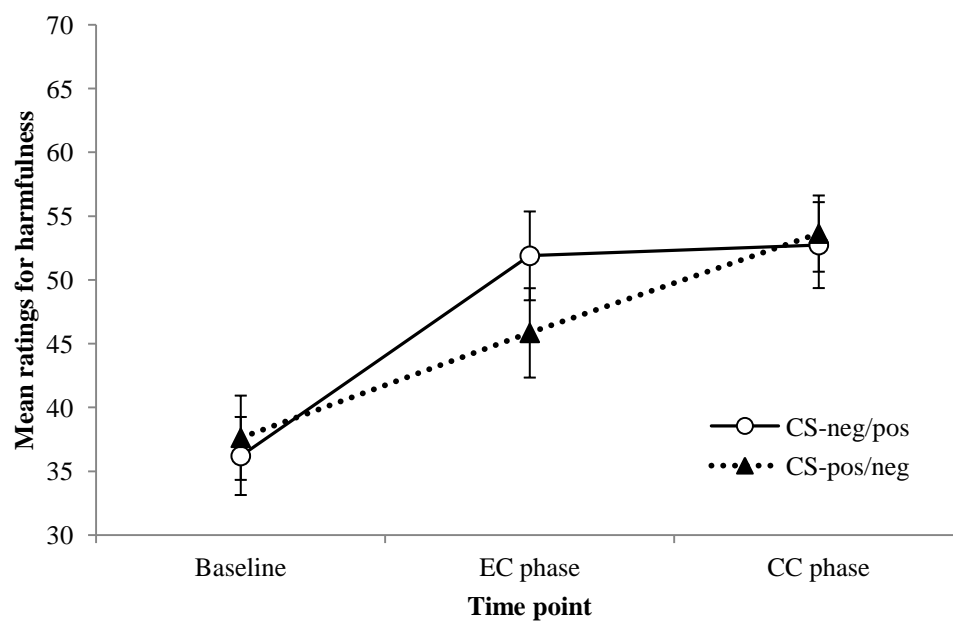


Figure 10: CS ratings for harmfulness during the experiment (higher values indicating more harmful ratings).

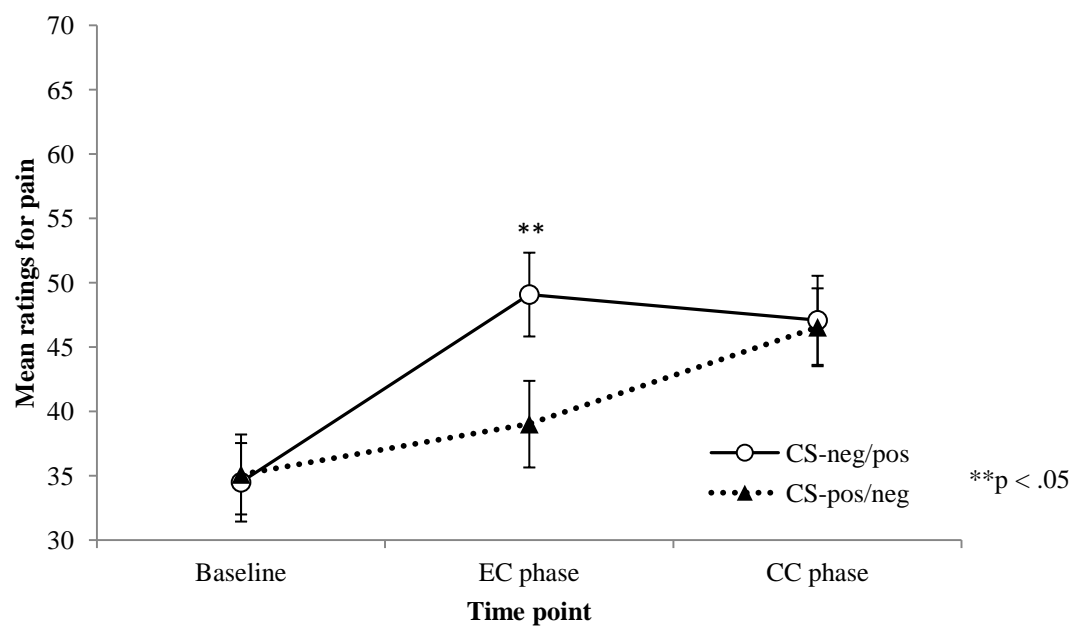


Figure 11: CS ratings for pain during the experiment (higher values indicating more painful ratings).

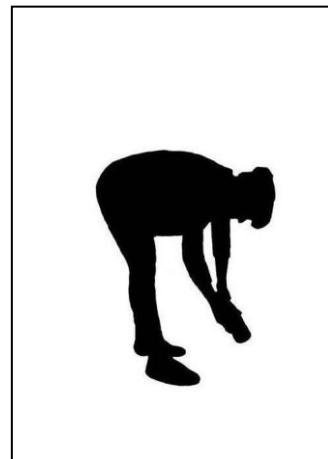
Table 1 Summary of differences and consistent results across studies.

	Differences between studies		Consistent results across studies
	Study 1	Study 2	
Sample characteristics	<ul style="list-style-type: none"> • $N = 33$ • <u>higher</u> depression levels than Study 2 ($t = 2.22, p < .05$) 	<ul style="list-style-type: none"> • $N = 50$ • <u>less</u> experience with CLBP than Study 1 ($\chi^2 = 7.18, p < .01$) 	<ul style="list-style-type: none"> • Samples from both studies comparable on <ul style="list-style-type: none"> - age and sex - pain-related questionnaire scores
Explicit results	<ul style="list-style-type: none"> • difference between CS-neg/pos and CS-pos/neg for valence after EC marginally significant ($p = .057$) 	<ul style="list-style-type: none"> • difference between CS-neg/pos and CS-pos/neg for valence after acquisition significant ($p < .01$) 	<ul style="list-style-type: none"> • course of ratings for all CS-neg/pos and CS-pos/neg • significant differences between CS-neg/pos and CS-pos/neg for pain (and valence) only after acquisition, not at baseline or after CC • trend to rate all CS as more negative, harmful and painful from baseline to CC
Implicit results	<ul style="list-style-type: none"> • 3-way interaction: shorter RT for congruent trials only for negative targets in APT-1 	<ul style="list-style-type: none"> • No RT x congruency x target valence interaction in APT-1 • main effect for target valence: shorter RT for positive targets 	<ul style="list-style-type: none"> • no main effect for congruency in either study

Notes: CLBP = chronic low back pain; CC = Counter Conditioning; RT = reaction times; APT-1 = first affective priming task.

Appendix A „Conditioned Stimuli“

Conditioned stimuli „Wiping“



Conditioned stimuli „Bending“

Appendix B: List of target words

Word list 1		Word list 2	
Positive	Negative	Positive	Negative
Liebe	Krieg	Freiheit	Alptraum
Paradies	Tod	Freude	Folter
Glück	Mord	Ferien	Gewalt
Sonne	Verrat	Freund	Tumor
Frieden	Tyrann	Leben	Seuche
Spaß	Sadismus	Herz	Sucht
kreativ	traurig	super	tot
beliebt	brutal	sonnig	einsam
warm	mies	vertraut	verboten
treu	korrupt	lieb	gemein
sinnlich	verlogen	mutig	schlimm
wertvoll	pervers	tolerant	asozial

A.2 Studie 2

Cross-cultural adaptation of the German Pain Solutions Questionnaire: an instrument to measure assimilative and accommodative coping in response to chronic pain

Robert Sielski¹, Julia A. Glombiewski¹, Winfried Rief¹, Geert Crombez², & Antonia Barke¹

¹Department for Clinical Psychology and Psychotherapy, University of Marburg

²Faculty of Psychology and Educational Sciences, Ghent University, Belgium

Corresponding author

Dr Antonia Barke
Philipps-University of Marburg
Department for Clinical Psychology and Psychotherapy
Gutenbergstraße 18
35032 Marburg, Germany
Email: abarke@gwdg.de
Phone: +49 6421 28-24045
Fax: +49 6421 28-28904

Acknowledgements

This study was supported by a grant from the German Research Foundation “Deutsche Forschungsgemeinschaft” DFG (Grant No. DFG-GL 607) to Julia A. Glombiewski. The DFG did not play any role in the design of the study and the collection, analysis and interpretation of the data. They did not participate in the writing of the manuscript or the decision to submit it for publication.

Conflicts of interest

The authors declare that they have no competing interests.

Abstract

According to the dual process model of coping, assimilative or accommodative strategies can be applied to deal with aversive life situations. In chronic pain, the tenacious focus on achieving analgesia is often referred to as assimilative coping; it is associated with more disability and catastrophic thinking. In contrast, accommodative coping (accepting one's pain and setting new goals) appears to have beneficial effects. To assess these different coping strategies in patients with chronic pain, questionnaires measuring these concepts are needed.

Following international guidelines, a German version of the Pain Solutions Questionnaire (PaSol) was prepared. A sample of 165 participants with chronic low back pain (60% women; age 53 ± 8.4) filled in the questionnaire and measures for pain-related disability, affective distress, catastrophic thinking, and attention to pain. Item analyses, an exploratory factor analysis, and correlations with pain-related measures were calculated. Additionally, data from 98 participants who received psychological treatment were examined to investigate the PaSol's sensitivity to change.

The exploratory factor analysis reproduced the original questionnaire's four-factor structure. Internal consistencies for the subscales were Cronbach's $\alpha = .72$ (Problem Solving), $\alpha = .75$ (Belief in a Solution), $\alpha = .81$ (Meaningfulness), and $\alpha = .84$ (Acceptance). Mean item difficulties for the subscales ranged from $p_i = .62$ (Belief in a Solution) to $p_i = .79$ (Solving Pain). The highest correlations were found for Meaningfulness with the Pain Catastrophizing Scale ($r = -.58$), the Hospital Anxiety and Depression Scale ($r = -.36$), and the Pain Disability Index ($r = -.30$). The PaSol subscale Meaningfulness predicted pain-related disability, and the subscales Meaningfulness and Solving Pain predicted affective distress, even after controlling for demographic and pain characteristics. Furthermore, the PaSol was found to be sensitive to detect changes over time.

The German version of the PaSol is a reliable and valid instrument in the measurement of assimilative and accommodative coping strategies in patients suffering from chronic low back pain. It may provide a useful tool when examining temporal dynamics of the changing coping strategies in the transition from acute to chronic pain as well as during pain treatments.

Key words: Coping, Questionnaire, Validation, Chronic pain

1. Background

Coping with chronic pain is a challenge for patients and healthcare providers. Patients suffering from chronic (low back) pain (CLBP) often express the wish for pain relief when beginning a new treatment. Most CLBP patients have tried several interventions to cure their pain or get more control over it ¹. The search for full recovery can involve great effort; despite this effort, the majority of attempts remain unsuccessful ²⁻⁴. Repeated failures in controlling pain can lead to less pain tolerance, more worry, and fear or catastrophic thinking, thus opening up new areas of concern ⁵⁻⁷.

A very useful model in this context is the dual process model of coping by Brandtstädter and Renner (1990) [7]. In this model, the authors distinguish two coping processes that can be activated when people are confronted with adverse life situations such as persistent pain. They call the tenacious search for a solution ‘assimilative coping’. Assimilative coping is often the first way of dealing with problems and can be successful if goal attainment is realistic and controllable. Regarding chronic pain, assimilative coping can mean focusing on achieving analgesia, such as through bed rest or taking (more) medication ⁹. If repeated attempts at achieving pain relief fail, chronic pain patients tend to intensify their efforts: often, they narrow their focus on, and increase the importance of, the goal of pain relief, rather than accepting the pain. As the probability of reaching the goal of a pain-free life is very low and achievement of this goal is not in the person’s control, the tenacity invested in further attempts can lead to adverse consequences in the long term ¹⁰⁻¹². Therefore, it may be more advantageous to change the coping strategy from assimilative to accommodative coping. ‘Accommodative coping’ means flexibly adjusting the goal when the initial goal cannot be reached, possibly aiming at a new goal. For chronic pain patients, this can mean relinquishing the goal of pain relief and setting new and realistic goals for a more satisfying life despite the pain. Acceptance of the insolubility of chronic pain has been found to be associated with lower reports of pain, less depression, and less disability ^{13,14}. However, the disengagement from pain is difficult, especially in the presence of catastrophic thinking and hypervigilance, which are often consequences of unsuccessful assimilative coping ¹⁵⁻¹⁷.

On the basis of the dual process model, the Pain Solutions Questionnaire (PaSol) ¹⁸ was developed to assess how people construct problems and seek solutions with regard to pain. In contrast to other instruments, the PaSol captures both the assimilative and accommodative ways of coping from an action-oriented and goal-dependent perspective. The PaSol is a 14-item instrument that aims at measuring different attitudes to solving the problem of pain by using four subscales. The subscales Problem Solving (ie, ‘I try everything to get rid of my pain.’) and Belief in a Solution (ie, ‘I am convinced that there is a treatment for my pain.’) represent the assimilative approach to deal with pain, whereas the subscales Acceptance of the Insolubility of Pain (ie, ‘I can accept that I can’t control my pain.’) and Meaningfulness of Life Despite Pain (ie, ‘I try to live with my pain.’) target

accommodative coping. The item construction aimed at ensuring that the problem-solving attitudes were particularly related to pain, both acute and chronic situations were presented, and potential outcome and process measures (ie, disability, attention) were kept separate. In order to create the items, items from the Tenacious Goal Pursuit and Flexible Goal Adjustment scale ⁸, the Dutch version of the Chronic Pain Acceptance Questionnaire (CPAQ) ¹⁹, and the Illness Cognition Questionnaire (ICQ) ²⁰ were inspected and, if they appeared useful within the context of the dual process model, adapted for the purpose at hand. The resulting scale was shown to have good reliability and validity. Cronbach's α coefficients for the subscales of the original scale range from $\alpha = .77$ to $\alpha = .86$ ^{7,18}. According to a content analysis of instruments that measure the construct of acceptance, ²¹ found the PaSol to be the only questionnaire besides the CPAQ that includes items on all relevant scales of acceptance (ie, disengagement from pain control, pain willingness, and engagement in activities other than pain control). Furthermore, assimilative coping as measured with the PaSol was shown to be related to disability, affective distress, attention to pain, and catastrophic thinking, and it explained unique variance in the prediction of medication-overuse headache ^{7,18,22}. To date, there is little research about assimilative and accommodative coping and its consequences for the specific group of patients suffering from CLBP.

To sum up: Despite indications that accommodative coping may have beneficial effects, research in this area and validated instruments for the assessment of these coping strategies are sparse. The purpose of the present study was to investigate the psychometric properties of the German adaptation of the PaSol in a CLBP sample and to provide further information about the factor structure and concurrent validity. In addition, we aimed at assessing whether the PaSol is sensitive to changes through a psychotherapeutic intervention.

2. Methods

2.1 Translation and cross-cultural adaptation

Permission to translate and validate the PaSol (English version, see ¹⁸) was obtained from the original authors. The translation and cross-cultural adaptation process followed the guidelines of Beaton and colleagues ²³. Prior to assessment, the pre-final version was given to a group of five CLBP patients. They filled in this version of the PaSol and provided general feedback about the questionnaire. Furthermore, they were interviewed about potential difficulties in understanding the items. The pre-final German and the back-translated versions of the questionnaire were also sent to the original authors of the PaSol, who approved the changes that were made.

2.2 Participants

The study sample consisted of 165 patients suffering from CLBP (defined as back pain persisting for more than three months) ²⁴. The patients were recruited from an outpatient clinic (Psychotherapie Ambulanz Marburg, Germany) as well as from an inpatient rehabilitation centre (MediClin Klinik am Hahnberg, Germany). All patients (n = 98) from the outpatient clinic received a psychotherapeutic treatment (exposure treatment or cognitive-behavioural therapy, CBT) for 10–15 sessions. The CBT consisted of psychoeducation regarding pain, graded activity, relaxation training, and different cognitive interventions like attention shifting. The exposure treatment consisted of psychoeducation regarding pain, a fear hierarchy, and several exposure sessions (for further details, see: ²⁵). For these patients, data before therapy and upon its completion are available, allowing the assessment of the questionnaire's sensitivity to change. All participants provided informed consent to participate, and the study was approved by the Ethics Committee of the Department of Psychology, Philipps-University Marburg, Germany.

2.3 Instruments

Problem Solving. The PaSol consists of 14 items rated on a 7-point Likert scale, ranging from 0 (*not at all applicable*) to 6 (*highly applicable*). In the original, the items are subdivided into four interrelated scales: (1) Solving Pain Scale (4 items), (2) Meaningfulness of Life Despite Pain Scale (5 items), (3) Acceptance of the Insolubility of Pain Scale (3 items), and (4) Belief in a Solution Scale (2 items). The subscales (2) Meaningfulness of Life Despite Pain and (3) Acceptance of the Insolubility of Pain assess an accommodative way of coping with pain, whereas the subscales (1) Solving Pain and (4) Belief in a Solution capture assimilative coping.

Pain intensity. Average pain intensity over the last four weeks was assessed using an 11-point numeric rating scale (NRS) from 0 (*no pain*) to 10 (*pain at its worst*) from the German Pain Questionnaire (Deutscher Schmerzfragebogen, DSF) ²⁶.

Pain-related disability. The German version of the Pain Disability Index (PDI) ^{27,28} was used to measure pain-related disability. The PDI is a 7-item scale assessing the degree to which people experience interference in seven areas of daily life (family/home responsibilities, recreation, social activity, occupation, sexual behaviour, self-care, basic life activities) caused by their pain. Each area of activity is rated on an 11-point scale ranging from 0 (*no disability*) to 10 (*total disability*), with higher scores indicating higher pain-related disability. The PDI has very good reliability and validity ²⁹.

Attention to pain. The Pain Vigilance and Awareness Questionnaire (PVAQ) ³⁰ was used to measure attention to pain. The PVAQ consists of 16 items ranging from 0 (*never*) to 5 (*always*) that assess

awareness, consciousness, and vigilance regarding pain over the last two weeks. The PVAQ has shown to be a reliable and valid instrument ³¹.

Affective distress. The German version of the Hospital Anxiety and Depression Scale (HADS) ^{32,33} was used for identifying affective distress. The scale assesses depressive and anxious symptoms in the last seven days. The HADS consists of two subscales (Depression, Anxiety) with 7 items each. Items are answered on a 4-point scale with item-specific response categories. Both scales have a scoring range of 0–21. The total score of the HADS can also be used as an index of general affective distress. The reliability and validity have shown to be acceptable ³⁴.

Catastrophic thinking. The German version of the Pain Catastrophising Scale (PCS) ^{35,36} was used for the assessment of catastrophic thinking about pain. The PCS is a 13-item self-report instrument measuring pain-related catastrophising on three subscales: Rumination, Magnification, and Helplessness. Items are answered on a 5-point Likert scale from 0 (*not at all*) to 4 (*all the time*). The total score ranges from 0 to 52, with higher scores indicating higher levels of catastrophic thinking. Psychometric properties for the PCS have shown to be excellent ³⁷.

2.4 Statistical analysis

First, item analyses were performed to calculate mean item scores and standard deviations, item difficulties ³⁸, and corrected item–total correlations for each item. Furthermore, mean inter-item correlations, mean item difficulty, and internal consistency (standardised Cronbach's α) were determined for each subscale separately.

In order to analyze the factor structure of the PaSol, we decided to conduct an exploratory factor analysis. The Kaiser–Meyer–Olkin test of sampling adequacy and Bartlett's test of sphericity were conducted to determine whether the data were appropriate for factor analysis. On theoretical grounds (to keep in line with the original questionnaire), we decided to extract four factors. For the extraction, we used the maximum likelihood method and rotated the extracted factors orthogonally (varimax).

To analyze the concurrent validity, Pearson correlations between the scores of the PaSol subscales and related constructs (pain-related disability, attention to pain, affective distress, catastrophic thinking) were calculated. For sex and PaSol subscales, point biserial correlation coefficients were computed. Sex differences for age, average pain intensity, average pain quality, pain duration, PDI, and PaSol subscales were analyzed with independent *t*-tests.

To test for the unique contribution of the PaSol subscales in accounting for the variability in disability (PDI, criterion) and affective distress (HADS, criterion), hierarchical multiple regression analyses with blockwise forced entry were computed. In the first block, age, sex, and pain measures, ie pain

intensity, pain quality, and pain duration, were entered as predictor variables; in the second block, the subscales of the PaSol were entered to assess whether they explain an additional increment. Multicollinearity was assessed according to the recommendations of Menard (1995)³⁹ suggesting that tolerance values below .20 should be of concern.

In order to test whether the questionnaire is sensitive to changes following a psychotherapeutic intervention, patients who had received treatment were divided into two groups: patients whose PDI scores had improved by at least 30% after the treatment and patients whose scores had not done so⁴⁰. 2 x 2 mixed design analyses of variance with the between-subjects factor improvement (yes/no) and the within-subject factor time (pre-/post-treatment) and the PaSol subscales as dependent variables were conducted. HSD post hoc tests for unequal group size were used to analyze interaction effects further. As measures of effect size, Cohen's d and η^2 ⁴¹ are reported. The data were analyzed using Statistica version 10 (Statsoft Inc, Tulsa, USA) and SPSS Statistics 22⁴².

3. Results

3.1 Study sample

Data from a total of 165 patients (inpatient and outpatient sample; 60% female) were used to analyze the factor structure, construct validity, internal consistency, and item properties. Descriptive statistics for the samples are shown in Table 1. Regarding demographic and pain-related variables, ie, age, duration of pain, disability, and pain intensity, the present study sample is comparable to other populations of CLBP patients as well as to the study sample of the original validation of the PaSol^{18,43}.

3.2 Missing data and normality of score distribution

Regarding the PaSol, 0.04% of values were missing. The maximum of missing answers was one (item 3: *'I try to live with my pain.'*). According to the Kolmogorov–Smirnov test, all items were non-normally distributed (all p -values < 0.05); however, no item exceeded the critical values for kurtosis (>2) or skewness (>7), and no participant obtained the minimum (0) or maximum (84) possible scores.

3.3 Item analyses and internal consistency

Standard item analyses were performed. The item difficulties, which in the context of attitude measurement show the extent to which the items are endorsed by the respondents (lower values

indicate less agreement with the item), lay between $p_i = .35$ (item 9) and $p_i = .83$ (item 12). For the subscales, mean item difficulties were as follows: Solving Pain: $p_i = .79$; Meaningfulness of Life Despite Pain: $p_i = .64$; Acceptance of the Insolubility of Pain: $p_i = .40$; Belief in a Solution: $p_i = .62$. In the context of attitude measurement, item difficulties between .20 and .80 are considered desirable, as medium difficulties better differentiate between respondents and are of higher diagnostic value⁴⁴.

The internal consistencies for the subscales showed Cronbach's α values of $\alpha = .72$ (Problem Solving), $\alpha = .75$ (Belief in a Solution), $\alpha = .81$ (Meaningfulness), and $\alpha = .84$ (Acceptance). Removing any items did not substantially improve the internal consistency of the subscales. The item-total correlations ranged from $r_{itc} = .02$ (item 12) to $r_{itc} = .62$ (item 2). The subscale correlations ranged between $r = -.10$ and $r = .36$. (For details, see Table 2.)

3.4. Validity

3.4.1 Factor structure

Bartlett's test of sphericity was significant ($\chi^2 = 957.41$, $df = 91$, $p < .001$), and the value of Kaiser-Meyer-Olkin measure was 0.75, indicating that the sample was adequate and the data appropriate for factor analysis.

The exploratory factor analysis with the decision to extract four factors on theoretical grounds revealed a solution that reflected the original factors perfectly. The eigenvalues ranged between 1.45 and 2.50, accounting for 56.30% of the total variance, with a mean item communality of 0.56 ($SD = 0.18$). As items loaded on the same factors as reported in the original article, factor labels remained the same (For further details, see Table 3).

3.4.2 Correlations with pain parameters and pain-related constructs

The highest correlations with pain-related outcomes and pain quality at baseline were found for the subscale Meaningfulness of Life Despite Pain and catastrophic thinking ($r = -.58$, $p < .01$): Experiencing life as meaningful despite the pain is associated with lower levels of catastrophising. The subscale Solving Pain only showed (small) significant correlations with pain parameters such as intensity ($r = .21$, $p < .05$) and pain duration ($r = -.19$, $p < .05$): higher intensity and longer duration were linked to searching for a solution to the pain. The subscale Acceptance of the Insolubility of Pain correlated negatively with catastrophic thinking ($r = -.25$, $p < .01$) and attention to pain ($r = -.16$, $p < .05$): persons accepting the pain as unsolvable reported lower levels of catastrophising thoughts and

less attention to the pain. This was also the only PaSol subscale that seemed related to age ($r = .25, p < .01$). The subscale Belief in a Solution showed negative correlations with pain duration ($r = -.27, p < .01$), affective distress ($r = -.17, p < .05$), and catastrophic thinking ($r = -.21, p < .01$) (For further details, see Table 4).

3.5 Regression analyses for disability and distress

Two multiple hierarchical regression analyses with the criterion pain-related disability (PDI) and distress (HADS), respectively, were calculated to examine whether the PaSol subscales contributed uniquely to explaining their variance when controlling for age, sex, and pain characteristics. Assessment of multicollinearity revealed tolerances $>.85$ and variance inflation factors (VIF) < 1.2 , indicating that multicollinearity did not present a problem.

The analysis with pain-related disability as a dependent variable showed that no subscale of the PaSol was related to disability when controlling for demographic and pain characteristics (Table 5). Further analyses revealed that the subscale Meaningfulness was only related to less disability when pain duration was excluded from the analyses, and only age, sex, pain intensity, and pain quality were controlled for, indicating shared variance between pain duration and Meaningfulness (Table 6). The analysis with distress as a criterion revealed that the subscale Meaningfulness was associated with less distress, and the subscale Solving Pain with more distress, even after controlling for demographic and pain characteristics (Table 7).

3.6 Sensitivity to change

To assess whether the PaSol subscales were sensitive with regard to change (eg, improvements through treatment), we calculated 2 x 2 mixed design ANOVAs with the factors of improvement (yes/no) and time (pre/post) and the PaSol subscales as dependent variables. For the subscales Solving Pain ($F(1,64) = 0.73, p = .396$) and Belief ($F(1,64) = 0.75, p = 0.749$), no effects were observed. The subscale Acceptance showed a main effect for time ($F(1,64) = 7.95, p = .006, \eta^2 = .031$) and tendencies regarding a main effect for improvement ($F(1,64) = 2.74, p = .102, \eta^2 = .029$) and the interaction ($F(1,64) = 2.87, p = .095, \eta^2 = .011$). For the subscale Meaningfulness, the ANOVA revealed significant main effects for improvement ($F(1,64) = 10.51, p = .002, \eta^2 = .093$) and time ($F(1,64) = 10.40, p = .002, \eta^2 = .045$) and a significant interaction improvement x time ($F(1,64) = 5.49, p = .022, \eta^2 = .024$). For the results of the post hoc tests, see Figure 1.

4. Discussion

This is the first study developing and validating a German translation of the PaSol and investigating it in a sample of patients suffering from CLBP. The psychometric properties were satisfactory, and the exploratory factor analysis reflected the same four-factor structure and factor loadings comparable to those of the original version. The reliability and validity analyses demonstrated acceptable to good results, and the scale was sensitive to detect changes over time.

Item analysis and reliability. The internal consistencies for the subscales ranged from Cronbach's $\alpha = .71-.84$, which is in line with other results (¹⁸: $\alpha = .72-.84$; ⁷: $\alpha = .77-.86$). Taking into account that the PaSol is a very short instrument, consisting of 14 items in total with subscales of two to five items, these values are no reason for concern, since the brevity limits the internal consistency for statistical reasons. This is supported by the fact that excluding particular items does not result in a substantial improvement of the internal consistency.

Mean item difficulties for the subscales ranged from $p_i = .62$ for the subscale Belief in a Solution to $p_i = .79$ for the subscale Solving Pain as desired in the context of attitude measurement ⁴⁴.

To analyze the validity of the PaSol, an exploratory factor analysis was preferred, since only the original study provided information regarding the factor structure and its subscales. The analysis revealed an identical item–subscale classification within a four-factor solution for the German version of the PaSol compared to the original version of the questionnaire, indicating that the factor structure was reproduced in patients suffering from CLBP and in a German population.

One aim in the development of the PaSol was identifying factors beyond pain-related parameters that influence outcome and process measures. Therefore, items were constructed to separate potential outcome and process measures. The small to moderate correlations between the subscales of the PaSol and pain characteristics, pain-related disability, attention to pain, and affective distress indicate that the PaSol has good discriminant validity and does indeed measure aspects of adjusting to living with CLBP that go beyond those captured simply by the pain parameters. The strongest correlations were found for catastrophic thinking and the PaSol subscales Meaningfulness of Life Despite Pain and Acceptance of the Insolubility of Pain. The correlative results of our study support findings that the tenacious use of assimilative coping strategies is associated with higher levels of catastrophising and worry, whereas accommodative coping, like accepting pain and disengaging from the goal of analgesia, has been found to be beneficial regarding pain-related outcomes ^{11,16,45,46}. Yet, there is little data on the course of which coping styles are being used by chronic pain patients and should be further examined. For clinicians, it can be of interest to differentiate which way of coping a patient uses and leads to different therapeutic strategies.

In a further step, we investigated the unique contribution of the PaSol subscales in explaining pain-related disability and affective distress, which are both important outcomes in the field of chronic pain recommended for investigation by the Initiative on Methods, Measurement, and Pain Assessment in Clinical Trials (IMMPACT)⁴⁰. The results for affective distress showed that the subscales Meaningfulness and Solving Pain had a unique contribution in explaining variance, even when controlling for age and sex as demographic variables and pain intensity, pain quality, and pain duration as pain characteristics. Less affective distress was reported when patients valued their lives as more meaningful despite having chronic pain. The opposite was found for the subscale Solving Pain. This pattern is in line with the results of the original PaSol study as well as other studies investigating different coping styles^{7,18}. However, none of the PaSol subscales explained variance for pain-related disability uniquely when also accounting for demographic and all pain characteristics. This contrasts with the original study, in which the subscale Meaningfulness was found to explain unique variance with regard to disability (although at 3%, this was not much). However, we further investigated the prediction of pain-related disability and found the subscale Meaningfulness to provide a unique contribution only when pain duration was excluded from the analyses. In the literature, the influence of pain duration on the use of pain coping strategies was found to be more complex than first expected. Assimilative coping strategies tended to be used more by patients with acute pain and accommodative coping strategies by patients who had experienced pain for a long time⁷. Our study sample showed a large range regarding pain duration. Although all patients suffered from chronic — not acute — pain (defined as pain for more than three months), the pain duration differed strongly between patients (6 months – 50 years). The rather complex relationship between pain duration and the used coping strategy might explain our finding to some extent, even though pain duration did not influence the unique contribution for affective distress. Additional studies are needed to examine the temporal aspects of the use and change of coping strategies.

A first step in this direction was the analysis of the relationship between treatment improvements and coping strategies as measured by the PaSol. Our results indicated that patients who improved during the treatment scored more highly on the subscale Meaningfulness compared to patients who did not improve during treatment and compared to pretreatment. The same relationship (albeit only a tendency) was found for the subscale Acceptance. These results indicate that treatment improvements can lead to a higher use of accommodative coping strategies such as accepting rather than ‘fighting’ pain, and experiencing a meaningful life. These results agree with findings showing that acceptance-based therapies might lead to reductions in pain intensity and depression¹⁴. Furthermore, the results also suggest the PaSol is a useful and sensitive instrument to detect such changes during a therapeutic process, even though at this point this can only be said for

accommodative coping. Assimilative coping was not affected by treatment improvements in this study.

The current study has some limitations. First, we did not conduct a confirmatory factor analysis because the study sample was too small. In a further study, the factor structure should be investigated to examine the model fit with confirmatory methods. Several relevant pain-related outcomes were assessed, but due to the already large number of questionnaires, a specific acceptance-based questionnaire, eg the CPAQ ⁴⁷, could not be included and should be used in a further validation study. Additionally, only self-reported measures were employed; for future studies, it would be desirable to assess behaviour directly, eg with the Behavioural Avoidance Test – Back Pain for CLBP patients ⁴⁸, and to further examine the validity and sensitivity of the PaSol.

5. Conclusion

In summary, this study supports the use of the translated German version of the PaSol as a reliable and valid instrument for the assessment of accommodative and assimilative coping strategies in a sample of CLBP patients. The reported four-factor solution in the original version of the PaSol was also found in this German version. First analyses showed the unique contribution of the PaSol in the prediction of pain-related disability and affective distress. Additionally, our results support the PaSol — especially the subscales for accommodative coping — to be sensitive to detect changes following psychotherapeutic interventions. This may be of particular interest, because to date there is little knowledge on the temporal dynamics of coping strategy change and how assimilative and adaptive coping affect the development from acute to chronic pain.

1. Gore M, Sadosky A, Stacey BR, Tai K-S, Leslie D. The Burden of Chronic Low Back Pain. *Spine (Phila Pa 1976)*. 2012;37(11):E668-E677. doi:10.1097/BRS.0b013e318241e5de.
2. van Tulder MW, Koes BW, Metsemakers JF, Bouter LM. Chronic low back pain in primary care: a prospective study on the management and course. *Fam Pract*. 1998;15(2):126-132.
3. Costa CM, Maher CG, Hancock MJ, Mcauley JH, Herbert RD, Costa LOP. The prognosis of acute and persistent low-back pain: a meta-analysis. *CMAJ*. 2012;184(11):613-624. doi:10.1503/cmaj.120627.
4. Sanderson KB, Roditi D, George SZ, Atchison JW, Banou E, Robinson ME. Investigating patient expectations and treatment outcome in a chronic low back pain population. *J Pain Res*. 2012;5:15-22. doi:10.2147/JPR.S28636.
5. Hayes SC, Bisset RT, Korn Z, et al. The Impact of Acceptance Versus Control Rationales On Pain Tolerance. *Psychol Rec*. 1999;49:33-47.
6. Gutiérrez O, Luciano C, Rodríguez M, Fink BC. Comparison between an acceptance-based and a cognitive-control-based protocol for coping with pain. *Behav Ther*. 2004;35(4):767-783. doi:10.1016/S0005-7894(04)80019-4.
7. Crombez G, Eccleston C, Van Hamme G, De Vlieger P. Attempting to solve the problem of pain: A questionnaire study in acute and chronic pain patients. *Pain*. 2008;137(3):556-563. doi:10.1016/j.pain.2007.10.020.
8. Brandtstädter J, Renner G. Tenacious goal pursuit and flexible goal adjustment: Explication and age-related analysis of assimilative and accommodative strategies of coping. *Psychol Aging*. 1990;5(1):58-67. doi:10.1037/0882-7974.5.1.58.
9. Van Damme S, Crombez G, Eccleston C. Coping with pain: A motivational perspective. *Pain*. 2008;139(1):1-4. doi:10.1016/j.pain.2008.07.022.
10. Rusu AC, Hasenbring M. Multidimensional Pain Inventory derived classifications of chronic pain: Evidence for maladaptive pain-related coping within the dysfunctional group. *Pain*. 2008;134:80-90. doi:10.1016/j.pain.2007.03.031.
11. Aldrich S, Eccleston C, Crombez G. Worrying about chronic pain: vigilance to threat and misdirected problem solving. *Behav Res Ther*. 2000;38:457-470.
12. Eccleston C, Crombez G. Worry and chronic pain: A misdirected problem solving model. *Pain*. 2007;132(3):233-236. doi:10.1016/j.pain.2007.09.014.
13. McCracken LM, Carson JW, Eccleston C, Keefe FJ. Acceptance and change in the context of chronic pain. *Pain*. 2004;109(1-2):4-7. doi:10.1016/j.pain.2004.02.006.
14. Veehof MM, Oskam MJ, Schreurs KMG, Bohlmeijer ET. Acceptance-based interventions for the treatment of chronic pain: A systematic review and meta-analysis. *Pain*. 2011;152(3):533-542. doi:10.1016/j.pain.2010.11.002.
15. Crombez G, Van Damme S, Eccleston C. Hypervigilance to pain: An experimental and clinical analysis. *Pain*. 2005;116:4-7. doi:10.1016/j.pain.2005.03.035.
16. Van Damme S, Crombez G, Eccleston C. Retarded disengagement from pain cues : the effects of pain catastrophizing and pain expectancy. *Pain*. 2002;100:111-118.
17. Crombez G, Lauwerier E, Goubert L, Van Damme S. Goal pursuit in individuals with chronic pain: A personal project analysis. *Front Psychol*. 2016;7(JUN):1-9. doi:10.3389/fpsyg.2016.00966.
18. De Vlieger P, Bussche E Van Den, Eccleston C, Crombez G. Finding a solution to the problem of pain: Conceptual formulation and the development of the Pain Solutions Questionnaire (PaSol). *Pain*. 2006;123(3):285-293. doi:10.1016/j.pain.2006.03.005.
19. McCracken L, Vowles K, Eccleston C. Acceptance of chronic pain: component analysis and a

- revised assessment method. *Pain*. 2004;107(1-2):159-166.
20. Evers AWM, Kraaijmaat FW, van Lankveld W, Jongen PJH, Jacobs JWG, Bijlsma JWI. Beyond Unfavorable Thinking: The Illness Cognition Questionnaire for Chronic Diseases. *J Consult Clin Psychol*. 2001;69(6):1026-1036.
 21. Lauwerier E, Caes L, Van Damme S, Goubert L, Rosseel Y, Crombez G. Acceptance: What's in a name? A content analysis of acceptance instruments in individuals with chronic pain. *J Pain*. 2015;16(4):306-317. doi:10.1016/j.jpain.2015.01.001.
 22. Lauwerier E, Paemeleire K, Van Damme S, Goubert L, Crombez G. Medication use in patients with migraine and medication-overuse headache: The role of problem-solving and attitudes about pain medication. *Pain*. 2011;152(6):1334-1339. doi:10.1016/j.pain.2011.02.014.
 23. Beaton DE, Bombardier C, Guillemin F, Ferraz MB. Guidelines for the process of cross-cultural adaptation of self-report measures. *Spine (Phila Pa 1976)*. 2000;25(24):3186-3191. doi:10.1097/00007632-200012150-00014.
 24. Treede R, Rief W, Barke A, et al. A classification of chronic pain for ICD-11. *Pain*. 2015;156(6):1003-1007. doi:10.1097/j.pain.0000000000000160.
 25. Riecke J, Holzapfel S, Rief W, Glombiewski JA. Evaluation and implementation of graded in vivo exposure for chronic low back pain in a German outpatient setting: a study protocol of a randomized controlled trial. *Trials*. 2013;14:203. doi:10.1186/1745-6215-14-203.
 26. Casser, HR, Hüppe M, Kohlmann T, et al. Deutscher Schmerzfragebogen (DSF) und standardisierte Dokumentation mit KEDOQ-Schmerz - Auf dem Weg zur gemeinsamen Qualitätsentwicklung der Schmerztherapie [German pain questionnaire and standardized documentation with the KEDOQ-Schmerz. A way for qual. *Schmerz*. 2012;2:168-175.
 27. Pollard C. Preliminary validity study of the pain disability index. *Percept Mot Ski*. 1984;59:974.
 28. Dillmann U, Nilges P, Saile H et al. Behinderungseinschätzung bei chronischen Schmerzpatienten [Assessing disability in chronic pain patients]. *Schmerz*. 1994;8:100-110.
 29. Tait R, Chibnall J, Krause S. The Pain Disability Index: psychometric properties. *Pain*. 1990;40(2):171-182.
 30. McCracken LM. "Attention" to pain in persons with chronic pain: a behavioural approach. *Behav Ther*. 1997;28:271-284.
 31. Roelofs J, Peters ML, McCracken L, Vlaeyen JWS. The pain vigilance and awareness questionnaire (PVAQ): Further psychometric evaluation in fibromyalgia and other chronic pain syndromes. *Pain*. 2003;101(3):299-306. doi:10.1016/S0304-3959(02)00338-X.
 32. Zigmond A, Snaith R. The hospital anxiety and depression scale. *Br Med J*. 1986;292:344.
 33. Hinz A, Schwarz R, Herrmann C, Buss U, Snaith R. Hospital Anxiety and Depression Scale - Deutsche Version (HADS-D). *Diagnostica*. 2002;48:112-113.
 34. Bjelland I, Dahl A, Haug T, Neckelmann D. The validity of the Hospital Anxiety and Depression Scale. An updated literature review. *Psychosom Res*. 2002;52:69-77.
 35. Sullivan MJL, Bishop SR, Pivik J. The Pain Catastrophizing Scale: Development and validation. *Psychol Assess*. 1995;7(4):524-532. doi:http://dx.doi.org/10.1037/1040-3590.7.4.524.
 36. Meyer K, Sprotta H, Mannion AF. Cross-cultural adaptation, reliability, and validity of the German version of the Pain Catastrophizing Scale. *J Psychosom Res*. 2008;64(5):469-478. doi:http://dx.doi.org/10.1016/j.jpsychores.2007.12.004.
 37. Osman A, Barrios F, Gutierrez P, Kopper B, Merrifield T, Grittmann L. The Pain Catastrophizing Scale: further psychometric evaluation with adult samples. *J Behav Med*. 2000;23(4):351-365.
 38. Bortz J, Döring N. *Forschungsmethoden Und Evaluation*. 3rd editio.; 2003.
 39. Menard S. Applied logistic regression analysis. Sage university paper series on quantative

- applications in the social sciences. 1995:07-106.
40. Dworkin RH, Turk DC, Farrar JT, et al. Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. *Pain*. 2005;113:9-19.
 41. Levine TR, Hullett CR. Eta Squared, Partial Eta Squared, and Misreporting of Effect Size in Communication Research. *Hum Commun Res*. 2002;28(4):612-625. doi:10.1093/hcr/28.4.612.
 42. Corp. I. IBM SPSS Statistics for Windows, Version 22.0. 2013.
 43. Leeuw M, Goossens M, van Breukelen G, et al. Exposure in vivo versus operant graded activity in chronic low back pain patients: Results of a randomized controlled trial. *Pain*. 2008;138:192-207.
 44. Fisseni H-J. *Lehrbuch Der Psychologischen Diagnostik*. Göttingen: Hogrefe; 1997.
 45. Crombez G, Eccleston C, De Vlieger P, Van Damme S, De Clercq A. Is it better to have controlled and lost than never to have controlled at all? An experimental investigation of control over pain. *Pain*. 2008;137(3):631-639. doi:10.1016/j.pain.2007.10.028.
 46. Viane I, Crombez G, Eccleston C, et al. Acceptance of pain is an independent predictor of mental well-being in patients with chronic pain: Empirical evidence and reappraisal. *Pain*. 2003;106(1-2):65-72. doi:10.1016/S0304-3959(03)00291-4.
 47. McCracken LM, Vowles KE, Eccleston C. Acceptance of chronic pain: Component analysis and a revised assessment method. *Pain*. 2004;107(1-2):159-166. doi:10.1016/j.pain.2003.10.012.
 48. Holzapfel S, Riecke J, Rief W, Schneider J, Glombiewski JA. Development and Validation of the Behavioral Avoidance Test – Back pain (BAT-Back) for Patients with Chronic Low Back Pain. *Clin J Pain*. 2016;(JANUARY):1. doi:10.1097/AJP.0000000000000349.

Table 1. Means and standard deviations for age, pain parameters, disability, and cognitive and affective parameters

	Mean	SD
Age	53.0	8.4
Pain duration (years)	13.3	9.7
Pain intensity	5.8	1.9
Pain quality	4.7	2.6
Disability (PDI)	32.4	12.6
Attention to pain (PVAQ)	44.3	9.8
Affective distress (HADS)	20.1	6.2
Catastrophic thinking (PCS)	24.6	11.7

Table 2. Item means, standard deviations, and item difficulties

Item	M	SD	Difficulty
1	3.74	1.75	.62
2	3.45	1.67	.57
3	4.39	1.57	.73
4	2.65	1.94	.44
5	2.45	1.76	.41
6	3.63	1.68	.61
7	4.59	1.37	.76
8	4.75	1.32	.79
9	2.11	1.93	.35
10	4.65	1.35	.77
11	4.86	1.20	.81
12	4.96	1.38	.83
13	3.08	1.47	.51
14	3.72	1.81	.62

Table 1. Varimax-rotated factor loadings, eigenvalues, and explained variance for the extracted factors, mean inter-item correlations, and item–total correlations for the subscales as constituted by the factors

Item		ML	AC	SP	BS	Correlation with subscale
1	Even when I have severe pain, I still find my life meaningful.	.79				.65
2	Even when I have severe pain, I can see a way out.	.83				.74
3	I try to live with my pain.	.50	.42			.50
8	I try to make the best of my life, despite the pain.	.67				.66
13	I don't let the pain get in my way.	.48				.50
4	I can live with the idea that there is no solution for my pain.		.86			.76
5	I can accept that I can't control my pain.		.82			.70
9	I can accept that there is no solution for my pain.		.69			.66
7	I keep searching for ways to control my pain.			.59		.46
10	I try everything to get rid of my pain.			.74		.65
11	I keep searching for a solution for my pain.			.75		.61
12	I would do anything to be without pain.			.44		.33
6	I have confidence that they will find a solution for my pain.				.70	.60
14	I am convinced that there is a treatment for my pain.				.72	.60
Eigenvalue		2.50	2.20	1.74	1.45	-
Explained variance		17.85	15.68	12.43	10.34	
Internal consistency ^a		.81	.84	.72	.75	-
Mean inter-item correlation ^a		.65	.79	.63	.71	-

Notes: Factor loadings <.30 not shown; ML: Meaningfulness of Life Despite Pain; AC: Acceptance of the Insolubility of Pain; SP: Solving Pain; BS: Belief in a Solution; ^astandardised Cronbach's α : values for the subscales formed on the basis of the factors; the total explained variance is 56.30%.

Table 4. Correlations of the PaSol subscales with age, pain parameters, disability, and cognitive and affective parameters

	SP	ML	AC	BS
Age	.00	.12	.25**	.00
Pain duration (years)	-.19*	-.05	.15	-.27**
Pain intensity	.21*	-.05	-.11	-.04
Pain quality	-.05	.19*	.17	-.13
Disability (PDI)	.06	-.30***	-.11	-.10
Attention to pain (PVAQ)	.12	-.21**	-.16*	.01
Affective distress (HADS)	.05	-.36***	.03	-.17*
Catastrophic thinking (PCS)	.14	-.58***	-.25**	-.21**

SP: Solving Pain; ML: Meaningfulness of Life Despite Pain; AC: Acceptance of the Insolubility of Pain; BS: Belief in a Solution. * $p < .05$, ** $p < .01$, *** $p < .001$

Table 5. Results of the multiple hierarchical regression with forced blockwise entry. Criterion: pain-related disability, with demographic characteristics entered in the first, pain characteristics in the second, and PaSol subscales in the last block.

	B	SE B	β	ΔR^2
Step 1				.058*
Constant	41.288	8.245	-	
Age	-0.276	0.129	-.193*	
Sex	2.944	2.234	.119	
Step 2				.302***
Constant	28.508	8.273	-	
Age	-0.299	0.116	-.208*	
Sex	1.337	1.977	.054	
Pain Duration	0.220	0.101	.179*	
Pain Intensity	2.945	0.516	.460***	
Pain Quality	-0.673	0.371	-.144	
Step 3				.320 n.s.
Constant	30.569	9.682	-	
Age	-0.271	0.120	-.189*	
Sex	1.118	2.067	.045	
Pain Duration	0.193	0.107	.157	
Pain Intensity	2.944	0.534	.460***	
Pain Quality	-0.592	0.390	-.127	
PaSol SP	0.496	0.996	.042	
PaSol ML	-1.684	1.099	-.152	
PaSol AC	0.566	0.691	.077	
PaSol BS	-0.066	0.722	-.008	

Notes: * $p < .05$, *** $p < .001$, n.s. not significant.

PaSol: Pain Solutions Questionnaire; (SP: Subscale Solving Pain, ML: Subscale Meaningfulness of Life Despite Pain, AC: Subscale Acceptance of the Insolubility of Pain, BS: Subscale Belief in a Solution).

Table 6. Results of the multiple hierarchical regression with forced blockwise entry. Criterion: pain-related disability, with demographic characteristics entered in the first, pain characteristics *without pain-duration* in the second, and PaSol subscales in the last block.

	B	SE B	β	ΔR^2
Step 1				.038*
Constant	40.402	7.254	-	
Age	-0.237	0.118	-.158*	
Sex	2.789	2.013	.109	
Step 2				.281***
Constant	26.959	6.869	-	
Age	-0.220	0.103	-.146*	
Sex	1.035	1.770	.040	
Pain Intensity	3.174	0.466	.469***	
Pain Quality	-0.655	0.327	-.137*	
Step 3				.337*
Constant	33.712	8.002	-	
Age	-0.196	0.103	-.130	
Sex	0.253	1.800	.010	
Pain Intensity	3.127	0.460	.463***	
Pain Quality	-0.458	0.337	-.096	
PaSol SP	0.613	0.911	.048	
PaSol ML	-2.719	0.874	-.251**	
PaSol AC	0.484	0.577	.064	
PaSol BS	-0.251	0.621	-.031	

Notes: * $p < .05$, ** $p < .01$, *** $p < .001$

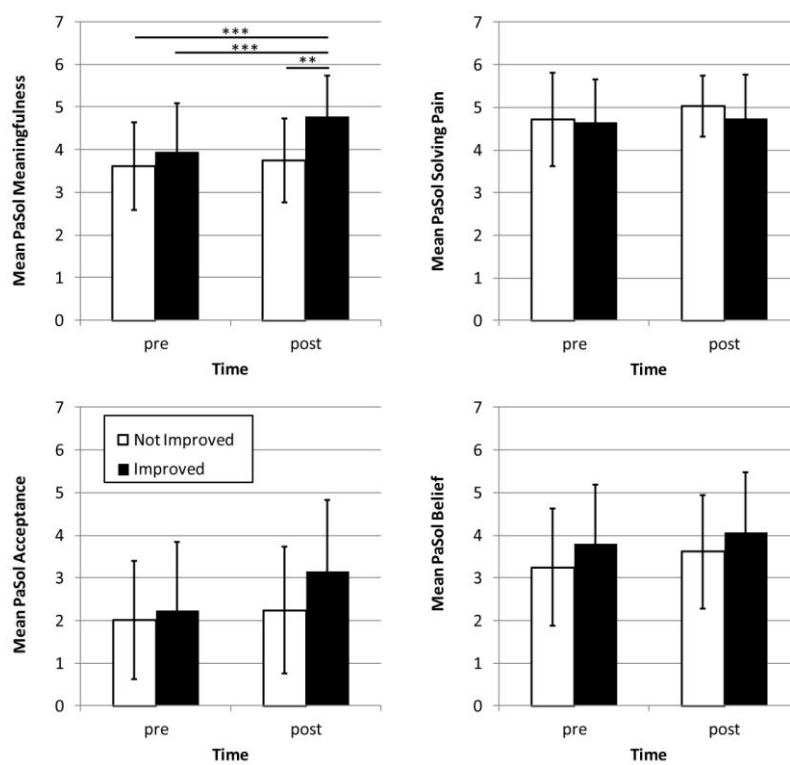
PaSol: Pain Solutions Questionnaire; (SP: Subscale Solving Pain, ML: Subscale Meaningfulness of Life Despite Pain, AC: Subscale Acceptance of the Insolubility of Pain, BS: Subscale Belief in a Solution).

Table 7. Results of the multiple hierarchical regression with forced blockwise entry. Criterion: affective distress, with demographic characteristics entered in the first, pain characteristics in the second, and PaSol subscales in the last block.

	B	SE B	β	ΔR^2
Step 1				.178
Constant	16.638	3.538	-	
Age	0.003	0.055	.005	
Sex	1.876	0.958	.179	
Step 2				.217
Constant	17.798	4.093	-	
Age	-0.014	0.058	-.023	
Sex	2.063	0.978	.197*	
Pain Duration	0.047	0.050	.090	
Pain Intensity	-0.209	0.255	-.077	
Pain Quality	0.001	0.183	.001	
Step 3				.392*
Constant	16.220	4.571	-	
Age	0.008	0.057	.014	
Sex	1.986	0.976	.190*	
Pain Duration	0.039	0.050	.075	
Pain Intensity	-0.247	0.252	-.091	
Pain Quality	0.095	0.184	.048	
PaSol SP	1.050	0.470	.212*	
PaSol ML	-1.685	0.519	-.359**	
PaSol AC	0.632	0.326	.202	
PaSol BS	0.160	0.341	.048	

Notes: * $p < .05$, ** $p < .01$

PaSol: Pain Solutions Questionnaire; (SP: Subscale Solving Pain, ML: Subscale Meaningfulness of Life Despite Pain, AC: Subscale Acceptance of the Insolubility of Pain, BS: Subscale Belief in a Solution).

Figure 1. Results of the post hoc tests for the changes in the PaSol subscales.

A.3 Studie 3

Int.J. Behav. Med.
DOI 10.1007/s12529-016-9572-9



Efficacy of Biofeedback in Chronic back Pain: a Meta-Analysis

Robert Sielski¹ · Winfried Rief¹ · Julia Anna Glombiewski¹

© International Society of Behavioral Medicine 2016

Abstract

Purpose The aims of the present analysis were to investigate the short- and long-term efficacy and treatment moderators of biofeedback as a psychological treatment option for chronic back pain.

Method A literature search using PubMed, PsycINFO, and the Cochrane Library identified 21 eligible studies including 23 treatment conditions and 1062 patients.

Results Meta-analytic integration resulted in a significant small-to-medium effect size for pain intensity reduction (Hedges' $g = 0.60$; 95 % confidence interval (CI) 0.44, 0.76) that proved to be stable with a significant small-to-large effect size (Hedges' $g = 0.62$; 95 % CI 0.40, 0.84) over an average follow-up phase of 8 months. Biofeedback also proved to be effective in reducing depression (Hedges' $g = 0.40$; 95 % CI 0.27, 0.52), disability (Hedges' $g = 0.49$; 95 % CI 0.34, 0.74), reduction of muscle tension (EMG; Hedges' $g = 0.44$; 95 % CI 0.22, 0.65), and improving cognitive coping (Hedges' $g = 0.41$; 95 % CI 0.26, 0.57). These effects remained comparatively stable at follow-up and for controlled studies only. Moderator analyses revealed longer biofeedback treatments to be more effective for reducing disability and a greater proportion of biofeedback in the treatment to be more effective for reducing depression. Publication bias analyses demonstrated the consistency of these effects.

Conclusion It is concluded that biofeedback treatment can lead to improvements on various pain-related outcomes in

the short and long terms, both as a standalone and as an adjunctive intervention.

Keywords Chronic back pain · Biofeedback · Psychological treatment · Meta-analysis

Introduction

Chronic pain is one of the major challenging health problems in Western societies. The (lower) back is the most common site for chronic pain [1–3], with a lifetime prevalence of 30 to 40 %. First episodes of low back pain have been reported for children and adolescents. The early onset of back pain has been found to be a significant predictor for chronic back pain in the adulthood [4, 5]. Individuals suffering from chronic back pain report substantial impairments in their daily activities, child care, social functioning, and work functioning, as well as lower overall quality of life [1, 6]. In addition to pain and disability, high muscle tension, low self-efficacy, and depression are common side effects of chronic back pain. Furthermore, chronic back pain is associated with high medical expenses, interferences with employment like work absenteeism, and disability, and therefore results in high socioeconomic costs [7–9]. Hence, it appears essential to identify effective and economical treatments for chronic back pain and associated impairments.

Psychological interventions have been shown to be effective in the treatment of chronic pain by reducing “pain, disability, psychological distress, and catastrophic ways of thinking” ([10], p.15). Henschke and colleagues [11] demonstrated in their systematic review that psychological treatments with cognitive behavioral elements are more effective than physical therapy, medication treatments, or back school in the short term, but there were no significant differences among specific psychological treatments (operant, cognitive,

✉ Robert Sielski
robert.sielski@staff.uni-marburg.de

¹ Department of Clinical Psychology and Psychotherapy,
Philipps-University of Marburg, Gutenbergstraße 18,
35032 Marburg, Germany

or respondent). Biofeedback, as a psychological treatment, is a very popular intervention among therapists and patients due to its combination of physiological and psychological methods. It is performed both as a standalone approach and as an additional element within cognitive behavioral therapy (CBT) or physical therapy. During biofeedback sessions, patients receive auditory, visual, or tactile feedback about physiological processes from their autonomous or central nervous system such as muscle tension, heart rate, or skin conductance. Biofeedback can be described as “operant conditioning of physiological activity” ([12], p. 35), by which “the patient learns to self-regulate his or her physiological processes with the help of feedback information” ([12], p. 36), and can comprise different sites, modalities, and procedures. There are various objectives biofeedback can target, e.g., developing more awareness or control for physiological processes and thus, consciously reducing muscle tension or influencing muscle imbalances. This is especially interesting in light of findings of higher baseline muscle activation and abnormal EMG responding to stress in chronic back pain patients [13–15]. But also, increases in self-efficacy and coping strategies can be aims of biofeedback treatment. Although, electromyographic (EMG) biofeedback is the most common used modality in the treatment of chronic pain, heart rate variability (HRV) or respiratory biofeedback, e.g., to support relaxation training and posture biofeedback, are also common. To date, there is no clear evidence of what is the primary mechanism of action for biofeedback in the treatment of chronic back pain. The beneficial effects of biofeedback have been demonstrated in pain conditions including chronic headache, temporomandibular disorders, and fibromyalgia [16–19].

Studies of biofeedback treatment in chronic back pain patients have shown inconsistent results. In a meta-analysis on psychological interventions for chronic low back pain (CLBP), Hoffman and colleagues [20] found that biofeedback was more effective than cognitive behavioral approaches in reducing depressive symptoms in CLBP patients. In addition, Flor and Birbaumer [21] found biofeedback treatment, relative to CBT and a waitlist control group, to be more effective at reducing pain severity and producing changes on affective, cognitive, and behavioral variables over the long term. Magnusson and colleagues [22] examined the effectiveness of postural biofeedback added to a conventional physiotherapy treatment for CLBP; results indicated an advantage of the enhanced treatment condition at 6-month follow-up, but these results should be viewed with caution due to the small sample size ($n = 10$). However, in a randomized controlled trial with a highly disabled sample, Glombiewski and colleagues [14] compared the effectiveness of CBT, CBT enhanced with biofeedback, and a waitlist control condition, and observed comparable improvements on pain-related outcomes in the two treatment groups over both the short and long terms, while the waitlist control group did not significantly improve. Two other studies demonstrated little to no improvement in chronic back pain after biofeedback [23, 24].

It is difficult to draw firm conclusions based on extant studies due to variability in sample size and characteristics, biofeedback modality (EMG, postural, or respiratory), treatment conditions, and control groups. Some studies examined biofeedback as a standalone intervention, while others examined biofeedback as an additional feature in conventional treatments. Control groups have included waitlist control groups, CBT, and physiotherapy, while other studies have not included control groups.

Thus, the effectiveness of biofeedback in reducing the symptomatology of back pain patients remains unclear. We therefore conducted a meta-analysis of controlled and uncontrolled chronic back pain treatment studies that included biofeedback, to examine short-term and long-term effects of biofeedback on pain-related outcomes. This meta-analysis focuses on studies which report biofeedback treatments as a standalone intervention as well as part of any treatment with at least 25 % biofeedback intervention of the total treatment time. Secondly, given the methodological variability in existing studies, another aim of the present meta-analysis was to determine the specific efficacy of biofeedback compared to various different control groups. In addition, moderator analyses were conducted to identify potential moderators of treatment effects.

Methods

Search Procedure

The meta-analysis was conducted in accordance with the QUORUM guidelines, taking into account the recent updates to these guidelines (“PRISMA guidelines” [25]). Studies were identified by searching PubMed, PsycINFO, and the Cochrane Library using the search term *biofeedback* combined with the term *back pain*. Studies published between the first available year and April 14, 2014, were included in the meta-analysis. In addition, reference lists from relevant studies and review papers identified in the database searches were manually reviewed. It was determined a priori that only published studies would be included. These search procedures identified 412 unique articles, which were then further examined by two independent reviewers (RS and JAG) for potential inclusion in the meta-analysis.

Determination of Outcome Variables

The Initiative on Methods, Measurement and Pain Assessment in Clinical Trials (IMMPACT) [26, 27] recommends the inclusion of a set of core outcome domains (e.g., pain, physical functioning, emotional functioning, symptoms,

and adverse events) and supplemental outcome domains (e.g., coping, interpersonal functioning) in clinical trials of pain treatments. Following these recommendations, we included average pain intensity as a primary outcome [28] and as a measure of the core outcome domain “pain.” Other outcome measures included the following: disability, as a measure of the suggested core outcome domain “physical functioning”; depression or another affective state (if depression was not assessed), as a measure of the core outcome domain “emotional functioning”; self-efficacy or coping (subsequently referred to as cognitive coping), as a measure of the supplemental outcome domain “coping”; and reduction of muscle tension (EMG), as an additional outcome, since pain is often associated with muscle tenseness.

We also examined biofeedback treatment dose, proportion of treatment time spent on biofeedback, sample size, and methodological quality of the studies as potential moderators.

Study Selection

Inclusion criteria for studies were as follows:

1. Study included patients with chronic back pain (in any region of the back)
2. Study included an adult sample (age 18 or older)
3. Study employed a biofeedback intervention of any kind for at least 25 % of the total treatment time
4. Study reported measures of at least one of the main outcome variables (see above) at both pre- and post-intervention or pre-intervention and follow-up
5. Study provided sufficient data to perform effect size analyses

If available, follow-up data (from the longest available follow-up) and data for control groups were included. Publications in English and German were considered.

Studies meeting the following criteria were excluded:

1. The study was a case study.

Validity Assessment

No additional methodological criteria were applied, and the meta-analysis included randomized controlled trials (RCTs) as well as uncontrolled or nonrandomized studies. However, to allow for comparison of effect sizes for RCTs and less methodologically sound studies and to control for confounding effects of study quality on effect size [30], we rated the quality of each study on a validity scale and analyzed this quality score as a moderator of the study findings. The validity scale was developed in a previous study by one of the authors (JAG,

[14])¹ by adapting Jadad criteria for pharmacological trials [31] and following PRISMA recommendations [25]. The validity scale includes aspects of internal, external, and construct validity and includes 20 dichotomous items, with a maximum score of 20. For each study, validity was assessed independently by two reviewers (JAG and RS) and inter-rater reliability was calculated. Disagreements were resolved through discussion.

Data Extraction

For each study, two of the authors (JAG and RS) independently selected psychometrically validated measures of pain intensity, depression, disability, cognitive coping, and reduction of muscle tension (EMG); these authors also extracted numerical data for analysis of changes from pre- to post-treatment and from pre-treatment to follow-up. Numerical data from 50 % of the studies was double checked by two of the authors (JAG and RS) to ensure reliability. Cohen's kappa was calculated for categorical items, and intraclass correlations (ICCs) were used for items measured on an interval scale. Cohen's kappa was 0.96 (95 % CI 0.95–0.97). All variables had significant ICCs of 0.97 or higher, with the exception of dropout rates. Differences were discussed and clarified.

Quantitative Data Synthesis

All analyses were completed manually or using the software program Comprehensive Meta-Analysis, version 2 [32]. Intention-to-treat (ITT) data were analyzed when available; when ITT data were not available, data from study completers were included. We calculated separate effect sizes for continuous measures of pain intensity, depression, disability, cognitive coping, and reduction of muscle tension (EMG) using within-group pre-post treatment differences for all studies and also for the group of controlled studies. Effect sizes were calculated using Hedges' *g*, a variation of Cohen's *d* that corrects for biases due to small sample sizes [33], and its 95 % confidence interval. Within-group effect sizes were calculated using the following formula:

$$d = \left(\frac{Y_1 - Y_2}{S_{\text{Difference}}} \right) \sqrt{2(1-r)},$$

where Y_1 is the pre-treatment sample mean, Y_2 is the post-treatment sample mean, $S_{\text{Difference}}$ is the standard deviation of the difference, and r is the correlation between pre-treatment

¹ The full version of the validity scale is available upon request from one of the authors (JAG).

and post-treatment scores. Hedges' g is computed by multiplying d by the correction factor

$$J(df) = 1 - \frac{3}{4df-1},$$

where df is the degrees of freedom to estimate the within-group standard deviation.

The effect sizes for controlled studies were computed using the following formula:

$$g = \frac{\Delta_{BFB} - \Delta_{CONT}}{\sqrt{\frac{(n_{BFB}-1)SD_{CONT}^2 + (n_{CONT}-1)SD_{BFB}^2}{(n_{total}-2)}}} \times \left(1 - \frac{3}{4(n_{BFB} + n_{CONT}) - 9}\right),$$

where Δ is the mean pre- to post-treatment change, SD is the standard deviation of post-treatment scores, n is the sample size, BFB refers to the treatment condition, and $CONT$ refers to the control condition.

The magnitude of Hedges' g can be interpreted using Cohen's [34] recommendations of small (0.2), medium (0.5), and large (0.8).

Although the correlation between pre- and post-treatment measures is needed in order to calculate the pre-post effect sizes, insufficient information on this correlation was included in the studies. We used a conservative estimate of $r = 0.7$, as recommended by Rosenthal [35].

Effect sizes for average pain intensity, depression, disability, cognitive coping, and reduction of muscle tension (EMG) were pooled across studies to obtain a summary statistic. It was decided a priori (based on previous pain research results) that effect sizes for individual studies greater than Hedges' $g = 3.0$ would be considered outliers and would be excluded from the analyses. No studies were determined to be outliers using this criterion. Effect size estimates were calculated using a random-effects model rather than a fixed-effects model because the studies were not functionally identical [36, 37]. Effect size estimates for follow-up data were also calculated in the manner described above.

Sensitivity Analysis

Publication bias may impact the results of a meta-analysis, as studies with nonsignificant results are less likely to be published than studies with significant results.

To address this potential for publication bias, we computed the *fail-safe* N [35], which indicates the number of studies that would be required to reduce the

overall effect size to a nonsignificant level. The *fail-safe* N was calculated using the following formula:

$$N = \frac{K(KZ^2 - 2.706)}{2.706},$$

where K is the number of studies in the meta-analysis and Z is the mean obtained from the K studies. The effect size can be considered robust if the number of studies (K) required to reduce the overall effect size to a nonsignificant level exceeds $5K + 10$ [35]. In addition, we constructed a funnel plot with the pre-post effect sizes for all outcomes and used the Trim and Fill method to examine the symmetry of the plot, which allowed us to determine whether negative or positive trials were over- or under-represented, accounting for the sample size. This information can then be used to re-calculate the effect size estimate.

Moderator Analysis

Four potential moderator variables were tested based on previous research. Quality of studies (assessed with a validity score), proportion of treatment time spent on biofeedback (relative to total treatment time), biofeedback treatment dose (total number of hours spent in biofeedback interventions), and sample size were chosen as potential moderators.

Moderating effects were examined using meta-regression analyses.

Results

Study Selection

The study selection process is shown in Fig. 1. Of the 412 potentially relevant articles identified in initial searches, 21 studies met all selection criteria. As noted above, no studies were excluded due to unusually high effect sizes ($g > 3.0$). These 21 studies included 23 treatment conditions and 1062 patients with back pain (see Tables 1 and 3). As required by the inclusion criteria, all 21 studies provided data for continuous measures of at least one relevant outcome variable at pre- and post-treatment. Eleven of the studies provided data at follow-up.

Characteristics of the Study Sample

Studies and Patient Characteristics

Table 1 provides information about the studies and treatment conditions included in the meta-analysis. Of the 21 studies included in our analysis, 18 used an EMG-based biofeedback

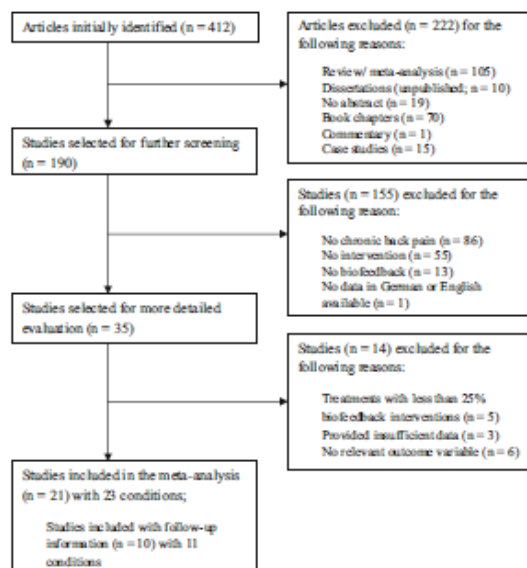


Fig. 1 Study selection process

training ($n = 921$), two ($n = 71$) used respiratory biofeedback, and one ($n = 70$) used postural biofeedback. Fourteen studies placed electrodes on participants' back muscles ($n = 776$), two studies placed electrodes on the front of the torso ($n = 54$), one study used a combination of back and front muscles ($n = 52$), and four studies did not report the placement of electrodes or did not use EMG-based biofeedback ($n = 180$). In eleven studies, the treatment consisted purely of biofeedback with no other intervention. The other studies combined biofeedback with another treatment, such as CBT, relaxation training, physical therapy, psychoeducation, or a combination of the above. These other interventions accounted for 33–75 % of the time study participants spent undergoing an intervention. The total number of minutes of biofeedback intervention ranged from 40 to 2856 ($M = 603$, $SD = 634$).

Five treatment conditions were uncontrolled or did not specify their control group ($k = 1$). Four control conditions consisted of CBT or operant-cognitive treatment, three consisted of physical therapy or waitlist control plus physical therapy ($k = 1$), four consisted of waitlist control, one consisted of relaxation training, two consisted of psychoeducation with or without placebo ($k = 1$), one consisted of a combination of education, physical therapy, relaxation, and psychological interventions, and one consisted of a placebo (noncontingent biofeedback). Because patients in waitlist control conditions (WLC) typically received treatment-as-usual (TAU), we merged studies employing a WLC condition with those employing a TAU control condition for the purpose of moderator analyses.

For 11 of the treatment conditions, follow-up data were reported, with follow-up periods ranging from 3 to 24 months ($M = 8.18$, $SD = 6.5$). The total number of patients across all studies was 1062, with 722 patients enrolled in treatment conditions and 340 patients in control conditions. The samples were predominantly female (65 % of patients). Twenty treatment conditions ($n = 716$ patients), and 16 control conditions ($n = 163$ patients) included sufficient data to compute dropout rates from pre- to post-treatment. A total of 121 patients (16.76 %) and 59 patients (17.35 %) dropped out of the treatment and control conditions, respectively, indicating comparable dropout rates for the treatment and control conditions.

Quality of Included Studies

The quality scores for each study are shown in Table 1. Scores ranged from 2 to 17 points (out of 20; $M = 10.48$, $SD = 4.35$). Two independent ratings of quality criteria were conducted; interrater reliability was $r = 0.98$. All 21 studies described their interventions sufficiently and defined adequate outcome measures. Eleven studies described dropout rates for each group. One study did not adequately describe inclusion and exclusion criteria. Twelve of the 21 studies implemented a manualized or otherwise standardized intervention.

Pre-Post Effect Sizes and Publication Bias

The pre-post effect sizes (Hedges' g) for pain intensity reduction (based on 21 studies with 23 conditions), depression (based on 11 studies), cognitive coping (based on 9 studies), disability (based on 14 studies), and reduction of muscle tension (EMG; based on 10 studies) are displayed in Table 2. All pre-post effect sizes were significant. According to Cohen's interpretation recommendations [34], the effect sizes for pain intensity reduction and disability were medium with confidence intervals suggesting small to medium effects. The effect sizes for depression, cognitive coping, and reduction of muscle tension (EMG) were small with confidence intervals suggesting small to medium effects. For the effect sizes for disability, depression, and cognitive coping, the Trim and Fill method indicated that the number of missing studies that would be needed to make the plot symmetrical was $n = 0$ studies, so all values remained unchanged. For the effect size for pain intensity, the Trim and Fill method indicated that $n = 4$ studies to the right of the mean would be needed to make the plot symmetrical. The adjusted value was Hedges' $g = 0.700$ (95 % CI 0.536–0.864). For the effect size for reduction of muscle tension (EMG), the Trim and Fill method indicated that $n = 1$ study to the right of the mean would be needed to make the plot symmetrical. The adjusted value was Hedges' $g = 0.479$ (95 % CI 0.268–0.689). The effect sizes for all outcomes for single studies are shown in Table 3.

Table 1 Characteristics of included studies

Authors	Year	Type of treatment	Description of intervention (N/N)	Placement of EMG electrode	Total number of minutes of biofeedback intervention	Type of cointervention (% time spent in cointervention)	Control conditions (N/N)	Longest follow-up in months	Measures	Quality score (x/20) ^a
Adams et al. [45]	1982	BFB	EMG (30/30)	Front	40–680	None	None	None	Pain (Analog Scale db) Disability n.m. Emot. Funct. n.m. Cognitive n.m.	2
Asfour et al. [46]	1990	BFB	EMG and CPRC (15/15)	Back	176	CPRC (75 % est.)	CPRC (15/15)	None	Muscle tension Pain (Level) Disability n.m. Emot. Funct. n.m. Cognitive n.m.	9
Donaldson et al. [38]	1994	BFB	SMUBT/EMG (12/12)	Back	350	None	Relaxation (12/12) Education (12/12)	3	Muscle tension n.m. Pain (MPQ) Disability n.m. Emot. Funct. n.m. Cognitive n.m.	16
Flor and Birbaumer [21]	1993	BFB	EMG (26/23)	Back	480	None	CBT (26/22)	24	Muscle tension Pain (MPQ) Disability (MPI) Emot. Funct. (MPI) Cognitive (Self efficacy)	16
Głombiewski et al. [14]	2010	BFB	EMG and CBT (62/52)	Back	540	CBT (60 %)	CBT (54/43)	6	Muscle tension Pain (Diary) Disability (PDI) Emot. Funct. (BDI) Cognitive (Coping) Muscle tension n.m.	16
Hallinan et al. [47]	2011	BFB	EMG (12/12)	Front	160	Paced breathing	WLC (12/11)	None	Pain (VAS) Disability (NDI) Emot. Funct. (HADS-D) Cognitive (SF-36 GH) Muscle tension n.m.	13
Huis in 't Veld et al. [48]	2010	BFB	EMG (82/52)	Back	2856 (est.)	None	None	None	Pain (VAS) Disability (PDI) Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	8
Kapiza et al. [42]	2010	BFB	Respiratory BFB (21/21)	Front	450	None	Noncontingent feedback (21/21)	3	Pain (VAS) Disability (PDI) Emot. Funct. (SCL-GSI) Cognitive n.m.	17
Keeffe et al. [49]	1981	BFB	EMG and Phys. (111/111)	Back	Not reported	Physical therapy, relaxation, education (67 % est.)	None	None	Muscle tension n.m. Pain (Pain Intensity) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension	3

Table 1 (continued)

Authors	Year	Type of treatment	Description of intervention (N/N)	Placement of EMG electrode	Total number of minutes of biofeedback intervention	Type of contraindication (% time spent in contraindication)	Control conditions (N/N)	Longest follow-up in months	Measures	Quality score (x/20)*
Kröner-Herwig and Beck [50]	2000	BFB	EMG (13/10)	Back	720	None	WCL (13/10)	None	Pain (Diary) Disability (NRS) Emot. Func. (NRS 0-10) Cognitive (Self-efficacy) Muscle tension	11
Kröner-Herwig and Beck (b) [50]	2000	BFB	EMG (13/10)	Back	720	None	WCL (13/10)	None	Disability (NRS) Emot. Func. (NRS 0-10) Cognitive (Self-efficacy) Muscle tension	11
Magnusson et al. [22]	2008	BFB	Postural BFB and Phys. (47/24 est.)	Back	150	Phys. (33 % est.)	Phys. (23/12 est.)	6	Pain (VAS) Disability (SF-36) Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	10
McLaughlin et al. [51]	2011	BFB	Respiratory (29/29)	Not reported	Not reported	Awareness training. Manual therapy (67 % est.)	None	None	Pain (NPRS) Disability (PSFS) Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	3
Neblett et al. [52]	2010	BFB	SEMGAS BFB (104/71)	Back	Not reported	Phys. (50 %)	Phys. (36/23)	None	Pain n.m. Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	4
Newton-John et al. [53]	1995	BFB	EMG (group of 4) (16/16)	Back	480	None	CBT (16/16)	6	Muscle tension Pain (Diary) Disability (PDI) Emot. Funct. (BDI) Cognitive (Self-efficacy) Muscle tension n.m.	14
Nouwen and Solinger [54]	1979	BFB	EMG (19/19)	Back	900	None	CG without specif. (7/7)	3	Pain (Report) Disability n.m. Emot. Funct. n.m. Cognitive n.m.	13
Nouwen [23]	1983	BFB	EMG (10/10)	Back	450	None	WCL (10/10)	None	Muscle tension Pain (Report) Disability n.m. Emot. Funct. n.m. Cognitive n.m.	10
De Sousa et al. [55]	2009	BFB	EMG and Phys. and Cog. (27/26)	Back and front	Not reported	Cognitive restriction techniques. Physical therapy (67 % est.)	WCL and physical treatment (25/18)	None	Pain (VAS) Disability (Roland-Morris DQ) Emot. Funct. (BDI) Cognitive n.m. Muscle tension n.m.	11

Table 1 (continued)

Authors	Year	Type of treatment	Description of intervention (N/N)	Placement of EMG electrode	Total number of minutes of biofeedback intervention	Type of intervention (% time spent in intervention)	Control conditions (N/N)	Longest follow-up in months	Measures	Quality score (x/20) ^a
Spence et al. [56]	1995	BFB	EMG (12/11)	Back	Not reported	None	WCL (12/11)	6	Pain (Index) Disability (WHYMPI Activity level) Emot. Funct. (BDI) Cognitive (Coping) Muscle tension n.m. Pain (Index) Disability (WHYMPI Activity level) Emot. Funct. (BDI) Cognitive (Coping) Muscle tension n.m.	13
Spence et al. [56]	1995	BFB	EMG and relaxation (12/9)	Back	Not reported	Relaxation (50 % est.)	WCL (12/11)	6	Pain (Index) Disability (WHYMPI Activity level) Emot. Funct. (BDI) Cognitive (Coping) Muscle tension n.m.	13
Strong et al. [57]	1989	BFB	EMG and relaxation (20/19)	Back	105	Relaxation (50 % est.)	Relaxation (20/18)	3–15	Pain (MPQ) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	8
Stuckey et al. [24]	1986	BFB	EMG (8/6)	Back	360	None	Placebo with education (8/6)	None	Pain (VAS) Disability n.m. Emot. Funct. n.m. Cognitive n.m. Muscle tension n.m.	9
Vlaeyen et al. [58]	1995	BFB	EMG and relax. (group) (21/13)	Not reported	990	Applied relaxation (50 % est.)	Operant-cognitive treatment (18/14)	12	Muscle tension Pain (VAS) Disability (Pain Impact) Emot. Funct. (Depression) Cognitive (Self efficacy) Muscle tension n.m.	11

N/W number of subjects, who began and completed the treatment (began/completed), db decibel, Muscle tension reduction of muscle tension (EMG), CPRC education, physical therapy, relaxation, psychological intervention, SM/UT single motor unit biofeedback training, MPQ McGill Pain Questionnaire, CBT cognitive behavioral therapy, MPI Multidimensional Pain Inventory, PD/Pain Disability Index, BDI Beck Depression Inventory, WLC wallist control, VAS visual analog scale, NDI Neck Disability Index, HADS-D Hospital Anxiety and Depression—Depression, SF-36 Short Form 36 Item Health Survey, SCL-GSI Symptom Check List—Global Severity Index, Phys physical therapy, NRS Numeric Rating Scale, NPGS Numeric Pain Rating Scale, PSFS Patient-Specific Functional Scale, SEMGAS BFB Surface EMG-assisted stretching biofeedback, CG control group, Cog. cognitive therapy, Roland-Morris Disability Questionnaire, WHYMPI West Haven-Yale Multidimensional Pain Inventory, Est. estimated values, n.m. not mentioned

^a Range [0–20] with a lower value indicating poorer quality of study

Table 2 Effect sizes for all outcome measures pre-post and pre-follow-up

Outcome	Type of effect	<i>k</i>	Hedges' <i>g</i>	95 % CI	<i>z</i>	<i>p</i> value	<i>I</i> ²	Fail-safe <i>N</i> (1/2tailed)
Pain intensity	Pre-post	22	0.601	0.439–0.763	7.29	<0.0001	77	1785/1251
Pain intensity	Pre-follow-up	11	0.623	0.404–0.841	5.59	<0.0001	67	360/251
Disability	Pre-post	14	0.542	0.339–0.744	5.25	<0.0001	79	608/424
Disability	Pre-follow-up	7	0.437	0.220–0.654	3.95	<0.0001	54	84/57
Emot. Funct.	Pre-post	11	0.398	0.272–0.524	6.20	<0.0001	27	205/141
Emot. Funct.	Pre-follow-up	6	0.486	0.145–0.826	2.80	0.005	78	69/47
Cognitive	Pre-post	9	0.414	0.261–0.567	5.32	<0.0001	36	142/97
Cognitive	Pre-follow-up	6	0.493	0.242–0.743	3.86	<0.0001	60	83/57
Muscle tension	Pre-post	10	0.438	0.221–0.654	3.97	<0.0001	75	239/165
Muscle tension	Pre-follow-up	3	0.309	0.032–0.585	2.19	0.029	44	7/4

Emot. Funct. = emotional functioning; Cognitive = cognitive Coping; Muscle tension = reduction of muscle tension (EMG); *I*² = heterogeneity statistics, values are percentages

The funnel plot for pain intensity is depicted in Fig. 2. The fail-safe *N*s are displayed in Table 2. These analyses suggest that the effect size estimates for all outcome variables were unbiased.

Effects at Follow-up

An effect size analysis from pre-intervention to the last available follow-up time point was conducted to examine the stability of biofeedback intervention effects (see Table 2). All follow-up effect sizes were small-to-medium and significant. For each effect size, except depression, the Trim and Fill method indicated that the number of missing studies that would be needed to make the plot symmetrical was *n* = 0 studies, so the Hedges' *g* values remained unchanged. For the effect size for depression, the Trim and Fill method indicated that *n* = 2 studies to the right of the mean would be needed to make the plot symmetrical. The adjusted value was Hedges' *g* = 0.618 (95 % CI 0.301–0.934). The fail-safe *N*s are displayed in Table 2. These analyses suggest that the effect size estimates for all outcome variables were unbiased.

Controlled Effect Sizes

For studies including control groups, we computed controlled effect sizes that compared the effectiveness of the intervention condition against the control condition. For reduction of muscle tension (EMG), the random-effects analysis of the controlled studies employing any control group comparison condition yielded a significant, medium mean effect size. For pain intensity, depression, and cognitive coping, the random-effects analysis of the

controlled studies yielded small but significant mean effect sizes (Hedges' *g*; see Table 4). The mean effect size for disability was not significant. Publication bias analyses suggested that the reported results are robust. These results should be considered preliminary given the small number of control conditions included in the analysis (range from *k* = 17 for the outcome of pain intensity to *k* = 6 for the outcome of reduction of muscle tension, EMG).

For studies including active control groups, the controlled effect sizes for biofeedback were similar in magnitude to those mentioned above. However, analyses of publication bias indicated that these effects were only robust for reduction of muscle tension (EMG) and pain intensity.

For studies including a wait list control group (range from *k* = 6 studies for pain intensity to *k* = 1 study for reduction of muscle tension, EMG), the controlled effect sizes for biofeedback were medium for depression and cognitive coping. The mean effect size for pain intensity was small, with confidence intervals suggesting small-to-big effect sizes. The mean effect sizes for disability and reduction of muscle tension (EMG) were not significant.

Moderator Analyses

To explore possible moderators of biofeedback treatment outcome, we examined study quality, proportion of treatment time spent on biofeedback, dose of treatment, and sample size in moderator analyses using only within-participants data from the treatment conditions. Results for each outcome measure are reported below.

Table 3 Effect sizes for all outcome measures for single studies

Author, publication year	Targeted symptom	Pre-post			Pre-follow-up		
		Hedges' <i>g</i>	95 % CI	<i>p</i>	Hedges' <i>g</i>	95 % CI	<i>p</i>
Adams et al., 1982	Pain	1.138	0.686–1.590	<0.0001	–	–	–
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	1.140	0.687–1.592	<0.0001	–	–	–
Asfour et al., 1990	Pain	0.454	0.062–0.845	0.023	–	–	–
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
Donaldson et al., 1994	Pain	0.582	0.136–1.027	0.011	0.943	0.441–1.444	<0.0001
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	0.319	–0.101–0.738	0.136	0.496	0.061–0.932	0.026
Flor and Birbaumer, 1993	Pain	1.440	1.022–1.859	<0.0001	1.365	0.933–1.797	<0.0001
	Disability	0.872	0.530–1.214	<0.0001	0.735	0.388–1.082	<0.0001
	Emot. Funct.	0.741	0.413–1.069	<0.0001	1.409	0.970–1.848	<0.0001
	Cognitive	0.589	0.275–0.903	<0.0001	0.975	0.600–1.351	<0.0001
	Muscle tension	0.187	–0.109–0.484	0.216	0.427	0.106–0.747	0.009
Glombiewski et al., 2010	Pain	0.329	0.134–0.525	0.001	0.308	0.080–0.536	0.008
	Disability	0.413	0.214–0.611	0.001	0.359	0.130–0.589	0.002
	Emot. Funct.	0.336	0.140–0.532	<0.0001	0.240	0.015–0.466	0.037
	Cognitive	0.588	0.382–0.795	<0.0001	0.625	0.381–0.868	<0.0001
	Muscle tension	–	–	–	–	–	–
Hallman et al., 2011	Pain	0.618	0.167–1.068	0.007	–	–	–
	Disability	0.735	0.268–1.202	0.002	–	–	–
	Emot. Funct.	0.320	–0.100–0.740	0.135	–	–	–
	Cognitive	0.105	–0.304–0.514	0.616	–	–	–
	Muscle tension	–	–	–	–	–	–
Huis in 't Veld et al., 2010	Pain	0.473	0.254–0.693	<0.0001	–	–	–
	Disability	0.353	0.139–0.566	0.001	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
Kapitza et al., 2010	Pain	0.469	0.132–0.806	0.006	0.674	0.319–1.030	<0.0001
	Disability	0.102	–0.218–0.421	0.533	–	–	–
	Emot. Funct.	0.352	0.023–0.681	0.036	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
Keefe et al., 1981	Pain	0.799	0.587–1.012	<0.0001	–	–	–
	Disability	–	–	–	–	–	–
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	0.337	0.147–0.527	<0.0001	–	–	–
Kröner-Herwig and Beck, 2000	Pain	0.293	–0.157–0.743	0.202	–	–	–
	Disability	0.693	0.195–1.191	0.006	–	–	–
	Emot. Funct.	0.456	–0.010–0.921	0.055	–	–	–
	Cognitive	0.295	–0.155–0.745	0.199	–	–	–
	Muscle tension	0.028	–0.411–0.467	0.901	–	–	–
Kröner-Herwig and Beck, 2000b	Pain	0.718	0.216–1.220	0.005	–	–	–
	Disability	0.661	0.168–1.154	0.009	–	–	–
	Emot. Funct.	0.194	–0.250–0.638	0.392	–	–	–
	Cognitive	0.803	0.286–1.320	0.002	–	–	–
	Muscle tension	0.058	–0.382–0.497	0.797	–	–	–
Magnusson et al., 2008	Pain	1.092	0.701–1.484	<0.0001	0.917	0.420–1.414	<0.0001
	Disability	0.000	–0.306–0.306	1.000	0.577	0.132–1.022	0.011
	Emot. Funct.	–	–	–	–	–	–
	Cognitive	–	–	–	–	–	–
	Muscle tension	–	–	–	–	–	–
McLaughlin et al., 2011	Pain	1.831	1.375–2.288	<0.0001	–	–	–
	Disability	1.410	1.017–1.802	<0.0001	–	–	–
	Emot. Funct.	–	–	–	–	–	–

Table 3 (continued)

Author, publication year	Targeted symptom	Pre-post			Pre-follow-up		
		Hedges' <i>g</i>	95 % CI	<i>p</i>	Hedges' <i>g</i>	95 % CI	<i>p</i>
Neblett et al., 2010	Cognitive	—	—	—	—	—	—
	Muscle tension	—	—	—	—	—	—
	Pain	—	—	—	—	—	—
	Emot. Funct.	—	—	—	—	—	—
	Cognitive	—	—	—	—	—	—
Newton-John et al., 1995	Disability	—	—	—	—	—	—
	Muscle tension	0.835	0.628–1.043	<0.0001	—	—	—
	Pain	0.718	0.309–1.127	0.001	0.712	0.211–1.213	0.005
	Disability	0.756	0.343–1.170	<0.0001	0.020	–0.419–0.459	0.929
	Emot. Funct.	0.677	0.274–1.081	0.001	0.451	–0.014–0.916	0.057
Nouwen and Solinger, 1979	Cognitive	0.356	–0.016–0.729	0.061	0.255	–0.192–0.703	0.263
	Muscle tension	—	—	—	—	—	—
	Pain	0.613	0.246–0.979	0.001	0.382	0.035–0.728	0.031
	Emot. Funct.	—	—	—	—	—	—
	Cognitive	—	—	—	—	—	—
Nouwen, 1983	Disability	—	—	—	—	—	—
	Muscle tension	0.578	0.215–0.940	0.002	0.049	–0.285–0.382	0.776
	Pain	0.150	–0.292–0.592	0.506	—	—	—
	Emot. Funct.	—	—	—	—	—	—
	Cognitive	—	—	—	—	—	—
de Sousa et al., 2009	Disability	—	—	—	—	—	—
	Muscle tension	0.678	0.182–1.173	0.007	—	—	—
	Pain	0.530	0.202–0.858	0.002	—	—	—
	Disability	0.806	0.451–1.161	<0.0001	—	—	—
	Emot. Funct.	0.206	–0.104–0.515	0.192	—	—	—
Spence et al., 1995a	Cognitive	—	—	—	—	—	—
	Muscle tension	—	—	—	—	—	—
	Pain	0.277	–0.155–0.709	0.209	0.272	–0.195–0.739	0.254
	Disability	0.010	–0.413–0.432	0.964	0.143	–0.317–0.603	0.543
	Emot. Funct.	0.174	–0.252–0.600	0.424	0.255	–0.211–0.721	0.283
Spence et al., 1995	Cognitive	0.329	–0.107–0.765	0.139	0.393	–0.085–0.871	0.107
	Muscle tension	—	—	—	—	—	—
	Pain	0.366	–0.057–0.789	0.090	0.589	0.125–1.052	0.013
	Disability	0.098	–0.311–0.507	0.638	0.307	–0.127–0.741	0.166
	Emot. Funct.	0.207	–0.206–0.620	0.326	0.278	–0.154–0.710	0.207
Strong et al., 1989	Cognitive	0.512	0.074–0.949	0.022	0.517	0.063–0.972	0.026
	Muscle tension	—	—	—	—	—	—
	Pain	0.000	–0.421–0.421	1.000	0.840	0.206–1.475	0.009
	Disability	—	—	—	—	—	—
	Emot. Funct.	—	—	—	—	—	—
Stuckey et al., 1986	Cognitive	—	—	—	—	—	—
	Muscle tension	—	—	—	—	—	—
	Pain	0.322	–0.170–0.815	0.200	—	—	—
	Disability	—	—	—	—	—	—
	Emot. Funct.	—	—	—	—	—	—
Vlaeyen et al., 1995	Cognitive	—	—	—	—	—	—
	Muscle tension	0.169	–0.312–0.650	0.491	—	—	—
	Pain	0.159	–0.194–0.512	0.377	0.132	–0.264–0.528	0.514
	Disability	0.862	0.445–1.278	<0.0001	0.931	0.449–1.413	<0.0001
	Emot. Funct.	0.743	0.343–1.144	<0.0001	0.338	–0.068–0.745	0.103
	Cognitive	0.075	–0.277–0.426	0.677	0.086	–0.309–0.481	0.669
	Muscle tension	—	—	—	—	—	—

Pain Intensity

Hedges' *g* for pain intensity reduction was moderated by the quality of studies ($B = -0.028$, $SE = 0.008$, $p = 0.001$), with studies employing less rigorous methodology (i.e., lower validity scores) reporting greater effect sizes.

Depression

Hedges' *g* for depression was moderated by the proportion of biofeedback in the intervention ($B = 0.004$, $SE = 0.002$, $p = 0.05$), with studies employing higher proportions of biofeedback reporting greater effect sizes.

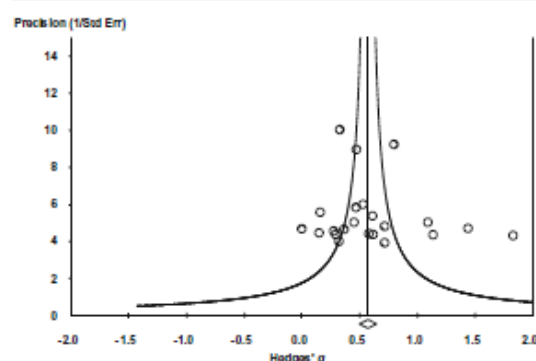


Fig. 2 Funnel plot of precision by Hedges' g for pre-post pain intensity measures

Cognitive Coping

Hedges' g for cognitive coping was moderated by the quality of studies ($B = -0.06$, $SE = 0.029$, $p = 0.031$), with studies employing less rigorous methodology reporting greater effect sizes.

Disability

Hedges' g for disability was moderated by the quality of studies ($B = -0.029$, $SE = 0.012$, $p = 0.016$), with studies with lower validity scores reporting greater effect sizes. The effect size for disability was moderated by dose of treatment ($B = 0.001$, $SE = 0.001$, $p = 0.004$), with studies employing larger dosages (more minutes of biofeedback training) reporting greater effect sizes.

Reduction of Muscle Tension (EMG)

Hedges' g for reduction of muscle tension (EMG) was moderated by the quality of studies ($B = -0.029$, $SE = 0.010$, $p = 0.003$), with studies with lower validity scores reporting greater effect sizes, and sample size ($B = 0.002$, $SE = 0.001$, $p < 0.05$), with studies with bigger sample sizes reporting greater effect sizes.

Sub-Analyses

Further analyses revealed that a pain intensity reduction of at least 30 % was reached in nine out of 22 studies (40.9 %) for

Table 4 Controlled effect sizes for all outcome measures

	k	Hedges' g	95 % CI	z	p	I^2	Fail-safe N (1/2tailed)
All controlled studies^a							
Pain	17	0.380	0.219–0.541	4.630	<0.0001	0	132/88
Disability	12	0.171	–0.006–0.349	1.892	0.059	0	9/3
Emot. Funct.	11	0.390	0.180–0.600	3.636	<0.0001	14	66/44
Cognitive	9	0.380	0.169–0.591	–3.527	<0.0001	0	41/26
Muscle tension	6	0.707	0.442–0.972	5.232	<0.0001	0	52/35
Active control groups^b							
Pain	11	0.362	0.171–0.552	3.720	<0.0001	6	56/36
Disability	7	0.133	–0.070–0.336	1.283	0.200	0	0/0
Emot. Funct.	6	0.260	0.042–0.478	2.337	0.019	0	9/5
Cognitive	4	0.269	0.014–0.525	–2.066	0.039	0	3/1
Muscle tension	5	0.694	0.416–0.971	4.896	<0.0001	0	37/25
CBT and education^c							
Pain	7	0.439	0.182–0.708	3.198	0.001	23	29/18
Disability	4	0.202	–0.096–0.500	1.327	0.184	20	0/0
Emot. Funct.	4	0.220	–0.036–0.475	1.685	0.092	0	2/0
Cognitive	4	0.269	0.014–0.525	–2.066	0.039	0	3/1
Muscle tension	3	0.495	0.080–0.909	2.341	0.019	0	5/2
Waitlist control^d							
Pain	6	0.465	0.127–0.804	2.692	0.007	0	11/6
Disability	5	0.297	–0.070–0.663	1.588	0.112	0	0/0
Emot. Funct.	5	0.690	0.242–1.138	3.017	0.003	29	20/13
Cognitive	5	0.619	0.244–0.994	–3.233	0.001	0	16/10
Muscle tension	1	0.839	–0.039–1.718	1.872	0.061	0	–

^a Emot. Funct. = emotional functioning; Cognitive = cognitive coping; Muscle tension = reduction of muscle tension (EMG); treatments that included biofeedback were tested against any control condition

^b Treatments that included biofeedback were tested only against active control groups

^c Treatments that included biofeedback were tested against CBT or interventions that used psychoeducation

^d Treatments that included biofeedback were tested against waitlist control conditions

treatments including at least 25 % biofeedback compared to two out of 18 studies (11.1 %) for control groups for pre-post data. At follow-up, the criterion of 30 % plus reduction was reached in seven out of 11 studies (63.3 %) for treatments with biofeedback and three out of eight studies (37.5 %) for control groups.

Correlational analyses were conducted to examine associations between effect sizes for the various outcome measures. For example, we examined the correlation between pain intensity reduction and reduction of muscle tension (EMG), as we anticipated a positive correlation, with lower EMG values associated with less pain. However, no significant correlations were found.

We also attempted to examine whether type of biofeedback treatment (EMG back vs. EMG front; EMG vs. respiration) was related to effect size. We hypothesized that a more specific biofeedback treatment (EMG back) would lead to more specific improvements in pain because it focuses on the region of the pain, while a different type of biofeedback (EMG front, respiration) would be expected to produce a more generalized relaxation. Unfortunately, no meaningful results can be reported because only a few studies used methods other than EMG-based biofeedback for the back, making the comparisons unbalanced.

Discussion

Summary of the Results

In this paper, we presented the results of a meta-analysis on the efficacy of biofeedback treatment for chronic back pain. The studies we analyzed included biofeedback training as at least 25 % of the intervention time and collected data on at least one of the following outcomes: pain intensity, disability, depression, cognitive coping, and reduction of muscle tension (EMG). First, within-participants analyses revealed significant, small-to-medium effect sizes for all reported outcomes at post-treatment. These results suggest that including biofeedback as one component of psychological or physiotherapeutic treatment seems to be helpful for chronic back pain patients on the outcomes highlighted by IMMPACT. Of note, analyses of publication bias suggest that these results can be considered robust. Secondly, findings were comparable when the subset of controlled studies was examined, with the exception of the effect size for pain reduction, which decreased from a medium effect size to a small effect size when including only controlled studies. Surprisingly, the effect sizes for all other outcomes remained relatively stable. Thirdly, the present results suggest that the effects of biofeedback treatment remained stable over long-term follow-up. The results suggest that cognitive behavioral therapy or physical therapy enhanced with biofeedback may lead to greater improvement in well-being compared to standard programs. Similarly, Hoffman and colleagues [20] demonstrated in their meta-analysis of psychological interventions for CLBP that psychological treatments

were “superior to wait-list control conditions in reducing pain ($p < 0.01$, $d = 0.48$)”, but different to the presented results, they could not show differences between CBT and self-regulatory treatments like biofeedback training for any outcome. The results of the current meta-analysis are in line with those of previous reviews (e.g., [11, 20]). Furthermore, Donaldson et al. [38] compared the effects of biofeedback training, relaxation training, and a psychoeducational group about CLBP on pain intensity and reduction of muscle tension (EMG). Their results showed that patients who received biofeedback training experienced an improvement in pain quality and intensity at follow-up compared to those who received relaxation training; effects of biofeedback did not significantly differ from effects of psychoeducation. These results suggest the importance of differentiating between relaxation and biofeedback training when examining effects of behavioral or respondent treatments. As the present meta-analysis used only studies that explicitly applied biofeedback training, our results suggest unique effects of (additional) biofeedback training, in line with Donaldson and colleagues’ study [38].

Overall, our meta-analysis of the existing literature suggests that biofeedback treatment, alone or in addition to other interventions, results in improvements in pain intensity, depression, cognitive coping, and reduction of muscle tension (EMG). Furthermore, moderator analyses indicated that longer biofeedback treatments were associated with greater reductions in pain-related disability. In addition, a greater proportion of biofeedback in the treatment was associated with larger effect sizes for reductions in depression.

However, the present results also show that despite high patient acceptance biofeedback treatment, the existing research is sparse. This is particularly true of methodologically rigorous studies, e.g., studies with control groups or studies assessing follow-up data. Despite the robust results, the results for long-term follow-up effects should be regarded with particular caution due to the small number of studies. In addition, due to the small numbers of studies and participants, it is not clear whether the statistically significant improvements also represent clinically significant improvements.

Strengths of the Current Study and Comparison with Previous Reviews

A strength of the present study is that we followed the methodological standards for conducting and reporting meta-analyses used by the Cochrane Reviews on chronic pain and recently recommended by QUORUM [10, 11, 25]. In addition, we adopted the IMMPACT criteria on outcome domains as encouraged by Morley et al. [39]. Previous reviews have focused on randomized controlled trials only (e.g., Cochrane Reviews; [40]); thus, some studies that were included in the present meta-analysis were omitted from previous reviews. To address this problem, we conducted moderator analyses, computed controlled effect sizes, and used sensitivity analyses to

test for publication bias. As a result, we were able to include these additional studies and also add to the literature by providing an up-to-date review. Given the sparse research on biofeedback treatment for chronic back pain, this approach appears to be important to provide more information about the efficacy of these interventions. Nevertheless, our results are comparable with previous findings. Morley and colleagues [40] also found small-to-medium effect sizes for biofeedback treatment compared to waitlist control conditions with respect to pain experience (as intensity, sensation, or unpleasantness), mood, and cognitive coping. In contrast to the present study, Morley and colleagues [40] reported on chronic pain in general, whereas the present analyses focused on chronic back pain patients.² Furthermore, in most previous studies, biofeedback was grouped as part of behavioral or respondent therapy along with interventions such as relaxation training, leading to difficulties in determining the specificity of the results to biofeedback treatment. Hoffman and colleagues [20] found medium-to-large effect sizes for self-regulatory treatments such as biofeedback or relaxation training for pain intensity ($d = 0.75$) and depression ($d = 0.81$) compared to a waitlist control group. Although this study showed self-regulatory treatments to be effective, the results rely on only three to four studies, respectively, and it is difficult to determine whether biofeedback, relaxation training, or another self-regulatory treatment accounted for the effects. As the current meta-analysis included only studies with biofeedback elements, a further strength of the meta-analysis is the higher specificity compared to previous reviews or meta-analyses.

Limitations

As observed by Hofmann and Smits [41], a limitation of meta-analyses in general is that the results are highly influenced by the selection of inclusion criteria, the quality of the studies included, and the outcome measures selected, in addition to the authors' expectations about the effects. We decided a priori to only include published studies. To obtain sufficient data, we selected relatively liberal inclusion criteria, resulting in heterogeneous study quality and some studies of unsatisfactory quality. Therefore, we computed the quality of the included studies using a validity rating scale based on modified Jadad criteria [31]. Our analyses revealed that study quality moderated the results for pain intensity, disability, and reduction of muscle tension (EMG). These findings underscore the importance of the quality of studies and suggest that the low quality of some of the included

studies represents a limitation of the present meta-analysis. Nevertheless, with the exception of disability, the results for all outcome variables remained significant when only controlled studies were included in the analysis.

Even though the outcome measures of this meta-analysis were followed by the IMMPACT recommendations, not all relevant outcome variables in the field of chronic back pain could have been assessed. For the core outcome domain "physical functioning," we chose outcome measures for disability. Besides disability, the construct of pain interference is an often investigated outcome in the field of chronic pain. Roughly, it describes the pain-related disruption with daily activities, but regarding a review by Wilson [29], the concept of pain interference "is not used or defined consistently or exclusively" (p. 500). Additionally, the measurement of pain interference includes a broad range of variables like quality of life with pain, pain-related task interference, pain disability and depression or functional disability. Maybe it is due to this assumption that the included studies mostly focused on reporting disability and only a few included pain interference as an outcome. Therefore, the reported data only focuses on disability as impairment of physical, psychosocial, and functional factors, but could also be seen as a kind of pain interference.

A further limitation of this meta-analysis is the heterogeneity of definitions of biofeedback and back pain in the studies, resulting in various combinations of biofeedback treatment and back pain not localized to a specific region. Thus, we are able to describe the general effect of biofeedback on back pain, but cannot make specific recommendations as to which biofeedback modality is best for which kind of back pain. We attempted to address this problem by comparing different biofeedback modalities, but results were not interpretable due to a small number of studies overall and highly unbalanced comparisons. Thus, one important finding of this meta-analysis is that there are only a few studies on the effectiveness of biofeedback treatment on chronic back pain, and only four of these studies [14, 21, 38, 42] were of high methodological quality. For effect sizes at follow-up after acute treatment, results should be considered preliminary for most of the outcomes, as only half of the studies reported follow-up data. The pool of studies with follow-up data was especially small for the outcome of reduction of muscle tension (EMG), with only three studies reporting follow-up data. To approach the problem of heterogeneity, we used the random-effects model for effect size analyses. There were no outliers, and our sensitivity analyses showed only small changes in effect sizes in both directions after adjustment for pre-post effect sizes.

² Our rationale for excluding studies on other pain syndromes such as fibromyalgia or headache was that these disorders show different symptom patterns, e.g., higher muscle tension in CLBP patients compared to fibromyalgia patients [59], and usually show different treatment effect sizes [60].

Clinical and Scientific Implications

Chronic back pain is often associated with depression, low cognitive coping (e.g., low self-efficacy expectations), high muscular tension, and disability in daily life (e.g., work absenteeism). The current results indicate that biofeedback treatment, whether as standalone treatment or as an additional feature in a psychological or physical therapy, can lead to improvements in pain intensity, muscular tension, emotional state, and cognitive coping among chronic back pain patients in the short term as well as in the long term. This is noteworthy, as previous analyses of long-term treatment effects for chronic back pain have been unable to demonstrate significant improvements. Hoffman et al. [20] found long-term treatment benefits for disability (e.g., return to work) for combined psychological and multidisciplinary treatments compared to an active control group, but did not find significant long-term effects on any other outcome variable, e.g., pain intensity. There are significant concerns about the long-term efficacy of some medication treatments for chronic back pain; as Martell et al. ([43], p. 123) observed in their meta-analysis, "opioids are commonly prescribed for but may only be efficacious for short-term treatment for chronic back pain (<16 weeks)." Given these concerns as well as high prevalence rates (up to 56 %) for side effects such as medication abuse or addiction, longer-term solutions are urgently needed. Another notable result of the present meta-analysis is that, consistent with Hofmann and colleagues' [20] results, depression was reduced after acute treatment using biofeedback.

Clinicians should consider additional biofeedback treatment when treating patients with chronic back pain. Data could show that biofeedback is helpful in reducing a variety of pain-related symptoms. Thus, the low utilization of biofeedback as an intervention or therapy seems surprising. This discrepancy may be due to the fact that biofeedback not only requires expensive technology but also specific training to achieve satisfying effects. Biofeedback offers various possibilities for treatment, involving different sites, modalities (e.g., EMG, skin temperature, perspiration, heart rate), and procedures. Even if one modality is chosen, there is still a variety of dimensions to choose from, like "verbal instructions, focused attention, relaxation procedures, feedback, stress challenges, and motor skill learning" [38], p. 35]. Perhaps, this is a too high threshold for practitioners, especially against the background of the lack of clarity if the use of biofeedback justifies the additional expenses compared to more common CBT interventions. Patients and clinicians should keep in mind that the effects of biofeedback on various symptoms are small to medium. However, the possibility of long-term improvements suggests that it may be worthwhile for patients and clinicians to consider this treatment approach.

Unlike to literature about biofeedback treatments for headache which shows consistent findings that self-efficacy seems to be the main action of mechanism for biofeedback interventions, the data for chronic back pain is still unclear. The results for EMG-based measures for controlled studies show the greatest effect sizes in this meta-analysis. Therefore, it seems possible that for chronic back pain, another action of mechanism, e.g., reduction of muscle tension, is more important than self-efficacy. Further research should investigate in the action of mechanisms for biofeedback in chronic back pain using experimental designs and mediation analyses in treatment studies.

Scientifically, our study implies that more RCTs are needed in the field of biofeedback treatment for chronic back pain. Methodological quality of the studies was found to be a significant moderator for some outcome variables, e.g., pain intensity and reduction of muscle tension (EMG), indicating that better study quality resulted in smaller effects. RCTs are methodologically better performed and have higher data quality [44]; thus, more RCTs would allow for more confidence in the effects of biofeedback treatment. Additionally, an exact description of measures, participant flow, and procedure (which was missing in some of the included studies) should be regarded as essential for identifying important moderators or process variables. For example, Nestoriuc and Martin [16] found out that home training increased effects for biofeedback in migraine treatment up to 20 % compared to in-session biofeedback only. As the descriptions of the included studies for the current meta-analysis were often vague, we could not examine this variable as a moderator. The same problem applies to the exact back pain diagnosis. Researchers should be encouraged to provide detailed and accurate documentation of their studies on the basis of current standards. In addition, we recommend that further research on biofeedback treatment in chronic back pain, measure behavioral variables such as pain behavior or work absenteeism, to include another outcome dimension aside from questionnaires or self-ratings.

Conclusions

This is the first meta-analysis on the efficacy of biofeedback treatment for chronic back pain using the current standard recommendations to examine the following outcomes: pain intensity, reduction of muscle tension (EMG), depression, cognitive coping, and disability. The present results indicated that except for disability, (additional) biofeedback treatment led to improvements on all outcome measures in the short and long terms. Due to the sparse data and methodological flaws

of some of the included studies, these results should be regarded with caution, but suggest that biofeedback may be promising as a standalone or adjunctive intervention for chronic back pain.

Compliance with Ethical Standards

Funding The study was supported by a doctoral thesis scholarship from the University of Marburg.

Conflict of Interest The authors declare that they have no conflict of interest.

Ethical Approval For this type of study, formal consent is not required.

References

*References marked with an asterisk indicate studies included in the meta-analysis

- Breivik H, Collett B, Ventafridda V, Cohen R, Gallacher D. Survey of chronic pain in Europe: prevalence, impact on daily life, and treatment. *Eur J Pain*. 2006;10(4):287–333. doi:10.1016/j.ejpain.2005.06.009.
- Bouhassira D, Lantéri-Minet M, Attal N, Laurent B, Touboul C. Prevalence of chronic pain with neuropathic characteristics in the general population. *Pain*. 2008;136(3):380–7. doi:10.1016/j.pain.2007.08.013.
- Johannes CB, Le TK, Zhou X, Johnston J a, Dworkin RH. The prevalence of chronic pain in United States adults: results of an Internet-based survey. *J Pain*. 2010;11(11):1230–9. doi:10.1016/j.jpain.2010.07.002.
- Andersson GB. Epidemiological features of chronic low-back pain. *Lancet*. 1999;354(9178):581–5. doi:10.1016/S0140-6736(99)01312-4.
- Hestbaek L, Leboeuf-Yde C, Kyvik KO, Manniche C. The course of low back pain from adolescence to adulthood: eight-year follow-up of 9600 twins. *Spine (Phila Pa 1976)*. 2006;31(4):468–72. doi:10.1097/01.brs.0000199958.04073.d9.
- Wolff R, Clar C, Lerch C, Kleijnen J. Epidemiology of chronic non-malignant pain in Germany. *Schmerz*. 2011;25(1):26–44. doi:10.1007/s00482-010-1011-2.
- Maniadakis N, Gray A. The economic burden of back pain in the UK. *Pain*. 2000;84(1):95–103. doi:10.1016/S0304-3959(99)00187-6.
- Pfingsten M, Schöps P, Wille T, Terp L, Hildebrandt J. Classification of chronic pain. Quantification and grading with the Mainz pain staging system. *Schmerz*. 2000;14(1):10–7. doi:10.1007/s004820000060.
- Scholic SL, Hallner D, Wittenberg RH, Hasenbring MI, Rusu AC. The relationship between pain, disability, quality of life and cognitive-behavioural factors in chronic back pain. *Disabil Rehabil*. 2012;34(23):1993–2000. doi:10.3109/09638288.2012.667187.
- Williams A, Eccleston C, Morley S. Psychological therapies for the management of chronic pain (excluding headache) in adults (review). *Cochrane Database Syst Rev*. 2012;11:CD007407. doi:10.1002/14651858.CD007407.pub3.
- Henschke N, Ostelo R, van Tulder M, et al. Behavioural treatment for chronic low-back pain (review). *Cochrane Database Syst Rev*. 2010;7:CD002014. doi:10.1002/14651858.CD002014.pub3.
- Schwartz NM, Schwartz MS. Definitions of biofeedback and applied psychophysiology. In: Schwartz MS, Andrasik F, editors. *Biofeedback: a practitioner's guide*, vol. 3. New York: Guilford Press; 2003. p. 27–42.
- Burns JW. Arousal of negative emotions and symptom-specific reactivity in chronic low back pain patients. *Emotion*. 2006;6(2):309–19. doi:10.1037/1528-3542.6.2.309.
- *Glombiewski JA, Hartwich-Tersek J, Rief W. Two psychological interventions are effective in severely disabled, chronic back pain patients: a randomised controlled trial. *Int J Behav Med*. 2010;17(2):97–107. doi:10.1007/s12529-009-9070-4.
- Jacobs JV, Henry SM, Jones SL, Hitt JR, Bunn JY. A history of low back pain associates with altered electromyographic activation patterns in response to perturbations of standing balance. *J Neurophysiol*. 2011;106(5):2506–14. doi:10.1152/jn.00296.2011.
- Nestorciuc Y, Martin A. Efficacy of biofeedback for migraine: a meta-analysis. *Pain*. 2007;128(1–2):111–27. doi:10.1016/j.pain.2006.09.007.
- Nestorciuc Y, Rief W, Martin A. Meta-analysis of biofeedback for tension-type headache: efficacy, specificity, and treatment moderators. *J Consult Clin Psychol*. 2008;76(3):379–96. doi:10.1037/0022-006X.76.3.379.
- Glombiewski JA, Bernady K, Häuser W. Efficacy of EMG- and EEG-biofeedback in fibromyalgia syndrome: a meta-analysis and a systematic review of randomized controlled trials. *Evid Based Complement Alternat Med*. 2013;2013. doi:10.1155/2013/962741.
- Hassett AL, Radvanski DC, Vaschillo EG, et al. A pilot study of the efficacy of heart rate variability (HRV) biofeedback in patients with fibromyalgia. *Appl Psychophysiol Biofeedback*. 2007;32(1):1–10. doi:10.1007/s10484-006-9028-0.
- Hoffman BM, Papas RK, Chatkoff DK, Kerns RD. Meta-analysis of psychological interventions for chronic low back pain. *Health Psychol*. 2007;26(1):1–9. doi:10.1037/0278-6133.26.1.1.
- *Flor H, Birbaumer N. Comparison of the efficacy of electromyographic biofeedback, cognitive-behavioral therapy, and conservative medical interventions in the treatment of chronic musculoskeletal pain. *J Consult Clin Psychol*. 1993;61(4):653–8. <http://psycnet.apa.org/journals/ccp/61/4/653/>.
- *Magnusson ML, Chow DH, Diamandopoulos Z, Pope MH. Motor control learning in chronic low back pain. *Spine (Phila Pa 1976)*. 2008;33(16):E532–8. doi:10.1097/BRS.0b013e31817df89a.
- *Nouwen A. EMG biofeedback used to reduce standing levels of paraspinal muscle tension in chronic low back pain. *Pain*. 1983;17(4):353–60. doi:10.1016/0304-3959(83)90166-5.
- *Stuckey SJ, Jacobs A, Goldfarb J. EMG biofeedback training, relaxation training, and placebo for the relief of chronic back pain. *Percept Mot Skills*. 1986;63(3):1023–36. doi:10.2466/pms.1986.63.3.1023.
- Liberati A, Altman DG, Tetzlaff J, et al. Annals of internal medicine academia and clinic the PRISMA statement for reporting systematic reviews and meta-analyses of studies that evaluate health care interventions. *Ann Intern Med*. 2009;151(4):W65–94. doi:10.1371/journal.pmed.1000100.
- Turk DC, Dworkin RH, Allen RR, et al. Core outcome domains for chronic pain clinical trials: IMMPACT recommendations. *Pain*. 2003;106(3):337–45. doi:10.1016/j.pain.2003.08.001.
- Dworkin RH, Turk DC, Farrar JT, et al. Core outcome measures for chronic pain clinical trials: IMMPACT recommendations. *Pain*. 2005;113:9–19.
- Turk DC, Dworkin RH, McDermott MP, et al. Analyzing multiple endpoints in clinical trials of pain treatments: IMMPACT recommendations. *Pain*. 2008;139(3):485–93. doi:10.1016/j.pain.2008.06.025.

29. Wilson M. Integrating the concept of pain interference into pain management. *Pain Manag Nurs*. 2014;15(2):499–505. doi:10.1016/j.pmn.2011.06.004.
30. Glass GV. Primary, secondary, and meta-analysis of research. *Educ Res*. 1976;5(10):3–8. doi:10.3102/0013189X005010003.
31. Jadad AR, Moore RA, Carroll D, et al. Assessing the quality of reports of randomized clinical trials: is blinding necessary? *Control Clin Trials*. 1996;17(1):1–12. doi:10.1016/0197-2456(95)00134-4.
32. Borenstein M, Hedges L V, Higgins JPT, Rothstein HR. *Comprehensive meta-analysis* version. 2006.
33. Hedges LV, Olkin I. Nonparametric estimators of effect size in meta-analysis. *Psychol Bull*. 1984;96(3):573–80. doi:10.1037/0033-2909.96.3.573.
34. Cohen J. *Statistical power analysis for the behavioral sciences*, 2nd edn. Vol 2nd. 1988. doi:10.1234/12345678.
35. Rosenthal R. *Meta-analytic procedures for social research* (rev. Ed.). 1991.
36. Hedges LV, Vevea JL. Fixed- and random-effects models in meta-analysis. *Psychol Methods*. 1998;3(4):486–504. doi:10.1037/1082-989X.3.4.486.
37. Moses LE, Mosteller F, Buehler JH. Comparing results of large clinical trials to those of meta-analyses. *Stat Med*. 2002;21(6):793–800.
38. *Donaldson S, Romney D, Donaldson M, Skubick D. Randomized study of the application of single motor unit biofeedback training to chronic low back pain. *J Occup Rehabil*. 1994;4(1):23–37. doi:10.1007/BF02109994.
39. Morley S, Williams A, Eccleston C. Examining the evidence about psychological treatments for chronic pain: time for a paradigm shift? *Pain*. 2013;154(10):1929–31. doi:10.1016/j.pain.2013.05.049.
40. Morley S, Eccleston C, Williams A. Systematic review and meta-analysis of randomized controlled trials of cognitive behaviour therapy and behaviour therapy for chronic pain in adults, excluding headache. *Pain*. 1999;80(1–2):1–13. doi:10.1016/S0304-3959(98)00255-3.
41. Hofmann SG, Smits JAJ. Pitfalls of meta-analyses. *J Nerv Ment Dis*. 2008;196:716–717.
42. *Kapitza KP, Passie T, Bemeck M, Karst M. First non-contingent respiratory biofeedback placebo versus contingent biofeedback in patients with chronic low back pain: a randomized, controlled, double-blind trial. *Appl Psychophysiol Biofeedback* 2010;35(3): 207–17. doi:10.1007/s10484-010-9130-1.
43. Martell BA, O'Connor PG, Kerns RD, et al. Systematic review: opioid treatment for chronic back pain: prevalence, efficacy, and association with addiction. *Ann Intern Med*. 2007;146(2):116–27. doi:10.7326/0003-4819-146-2-200701160-00006.
44. Guyatt G, Oxman AD, Akl EA, et al. GRADE guidelines: 1. Introduction - GRADE evidence profiles and summary of findings tables. *J Clin Epidemiol*. 2011;64(4):383–94. doi:10.1016/j.jclinepi.2010.04.026.
45. *Adams J, Pearson SJ, Olson N. Innovative cross-modal technique of pain intensity assessment with lower back pain patients given biofeedback training. *Am J Clin Biofeedback* 1982;5(1):25–30.
46. *Asfour SS, Khalil TM, Waly SM, Goldberg ML, Rosomoff RS, Rosomoff HL. Biofeedback in back muscle strengthening. *Spine (Phila Pa 1976)*. 1990;15(6):510–13.
47. *Hallman DM, Olsson EMG, von Schéele B, Melin L, Lyskov E. Effects of heart rate variability biofeedback in subjects with stress-related chronic neck pain: a pilot study. *Appl Psychophysiol Biofeedback* 2011;36(2):71–80. doi:10.1007/s10484-011-9147-0.
48. *Huis in't Veld, RMH, Kosterink SM, Barbe T, Lindegård A, Marecek T, Vollenbroek-Hutten MMR. Relation between patient satisfaction, compliance and the clinical benefit of a tele-treatment application for chronic pain. *J Telemed Telecare*. 2010;16(6):322–8. doi:10.1258/jtt.2010.006006.
49. *Keefe FJ, Block AR, Williams RB, Surwit RS. Behavioral treatment of chronic low back pain: clinical outcome and individual differences in pain relief. *Pain* 1981;11(2):221–31.
50. *Kröner-Herwig B, Beck A. An exploratory study of biofeedback for chronic low back pain. *Br J Ther Rehabil*. 2000;7:134–42.
51. *McLaughlin L, Goldsmith CH, Coleman K. Breathing evaluation and retraining as an adjunct to manual therapy. *Man Ther*. 2011;16(1):51–52. doi:10.1016/j.math.2010.08.006.
52. *Neblett R, Mayer TG, Brede E, Gatchel RJ. Correcting abnormal flexion-relaxation in chronic lumbar pain: responsiveness to a new biofeedback training protocol. *Clin J Pain*. 2010;26(5):403–9.
53. *Newton-John TR, Spence SH, Schotte D, Wing C, Mary S, Street P. Cognitive-behavioural therapy versus EMG biofeedback in the treatment of chronic low back pain. *Behav Res Ther*. 1995;33(6): 691–7. doi:10.1016/0005-7967(95)00008-L.
54. *Nouwen A, Solinger JW. The effectiveness of EMG biofeedback training in low back pain. *Biofeedback Self Regul*. 1979;4(2):103–11.
55. *Santaella da Fonseca Lopes de Sousa K, Garcia Orfale A, Mara Meireles S, Roberto Leite J, Natour J. Assessment of a biofeedback program to treat chronic low back pain. *J Musculoskelet Pain* 2009;17(4):369–77. doi:10.3109/10582450903284828.
56. *Spence SH, Sharpe L, Newton-John T, Champion D. Effect of EMG biofeedback compared to applied relaxation training with chronic, upper extremity cumulative trauma disorders. *Pain* 1995;63(2):199–206. doi:10.1016/0304-3959(95)00047-V.
57. *Strong J, Cramond T, Maas F. The effectiveness of relaxation techniques with patients who have chronic low back pain. *Occup Ther J Res*. 1989;9(3):184–92.
58. *Vlaeyen JW, Haazen IW, Schuerman JA, Kole-Snijders AM, van Eek H. Behavioural rehabilitation of chronic low back pain: comparison of an operant treatment, an operant-cognitive treatment and an operant-responder treatment. *Br J Clin Psychol*. 1995;34(Pt 1): 95–118.
59. Thieme K, Rose U, Pinkpank T, Spies C, Turk DC, Flor H. Psychophysiological responses in patients with fibromyalgia syndrome. *J Psychosom Res*. 2006;61(5):671–9. doi:10.1016/j.jpsychores.2006.07.004.
60. Malone MD, Strube MJ. Meta-analysis of non-medical treatments for chronic pain. *Pain*. 1988;34(3):231–44. doi:10.1016/0304-3959(88)90118-2.

A.4 Studie 4

COMMENTARY

For reprint orders, please contact: reprints@futuremedicine.com

Biofeedback as a psychological treatment option for chronic back pain



Robert Sielski¹ & Julia Anna Glombiewski^{*1}

First draft submitted: 29 August 2016; Accepted for publication: 10 October 2016;
Published online: 4 November 2016

Biofeedback as a psychological treatment option for chronic back pain was developed decades ago [1,2], and thus is a long-standing treatment option for chronic pain. However, for chronic low back pain, the most prevalent pain disorder, three questions about biofeedback remain unanswered:

- Is biofeedback an effective intervention for the treatment of chronic low back pain?
- What are the mechanisms of change?
- What are the limitations of biofeedback for chronic low back pain?

In this commentary, we will describe biofeedback for chronic low back pain and summarize current research on biofeedback to address these questions.

Biofeedback as an intervention for chronic low back pain

Early biofeedback approaches used simple auditory or tactile feedback stimuli. Modern biofeedback technologies are more

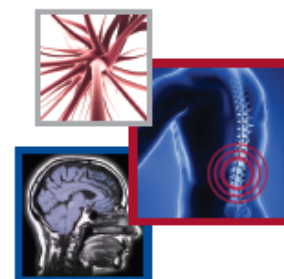
complex and have a variety of options for type of feedback, graphic design on the display and physiological reactions that may be targeted for feedback. Despite these technological advancements, the main principles of biofeedback have remained the same: to display physiological processes from the autonomic nervous system or CNS, such as muscular activity, and to provide feedback about these bodily reactions, which often occur outside of conscious awareness.

In the treatment of chronic back pain, biofeedback is often used as an adjunctive treatment option in a cognitive-behavioral therapy (CBT)-based treatment or as a supportive feature in relaxation trainings, but it is also used as a standalone intervention. The principle behind biofeedback has been described as operant learning: the patient learns to self-regulate physiological processes using the feedback provided [3].

Electromyography (EMG) based biofeedback is the most common biofeedback modality used in the treatment of chronic back pain. The major aim of

“...there is evidence that biofeedback is an effective psychological intervention for patients suffering from chronic back pain.”

Pain Management



KEYWORDS

- biofeedback • CBT • low back pain
- psychology

“Biofeedback as a psychological treatment option for chronic back pain was developed decades ago, and thus is a long-standing treatment option for chronic pain.”

¹Department of Clinical Psychology & Psychotherapy, Philipps-University of Marburg, Gutenbergstraße 18, D-35032 Marburg, Germany

^{*}Author for correspondence: Tel.: +49 0 6421 282 3617; Fax: +49 0 6421 282 8904; julia.glombiewski@uni-marburg.de

COMMENTARY Sielski & Glombiewski

“Although biofeedback ostensibly focuses on changes in physical processes, for example, the reduction of muscle tension in the lower back, the main treatment targets may be psychological rather than physiological.”

EMG biofeedback is to reduce muscle tension. However, a review of the literature indicates that several other biofeedback modalities have been examined in the treatment of chronic back pain, including respiratory biofeedback, postural biofeedback and surface EMG-assisted stretching biofeedback [4]. The feedback modality depends on the theoretical rationale and treatment objectives: respiratory biofeedback focuses on changing breathing patterns, that is, from a high breathing frequency to deep breathing, to support relaxation techniques and thus to improve well-being. Postural biofeedback aims to provide feedback on kinematic responses during body control learning, with the goal of modifying avoidance or overstraining of specific muscle groups in daily tasks. Postural biofeedback may also reveal muscle imbalances in resting postures. The goal of surface EMG-assisted stretching biofeedback is to reduce muscle activity and increase the range of motion and movement to a more healthy level [5–7].

Biofeedback in chronic low back pain is primarily a psychological or physiotherapeutic intervention, and is also helpful as a diagnostic tool. Clinicians can use biofeedback to establish whether patients are able to relax and assess patients' psychophysiological reactions to stressors. Understanding associations between emotions, stress, pain and physiological processes may boost patients' adherence to psychological pain treatments. Through biofeedback, patients learn about physiological processes and the ways in which biological and psychological factors can interact and lead to persistent pain complaints. Avoidance of feared movements can also be detected through biofeedback. One mechanism of biofeedback may be an increase in subjective sense of control over one's body and one's physical complaints, which may in turn lead to less catastrophic thinking and pain-related fear [3].

The above examples include only a few of the many possible modalities and treatment targets of biofeedback training.

Empirical support: is biofeedback a useful & effective intervention for the treatment of chronic back pain?

Analyzing empirical support for biofeedback in the treatment of chronic pain is challenging. There is a strong evidence for the efficacy of biofeedback in treatment of headaches and migraines [8,9]. For fibromyalgia, beneficial effects of biofeedback have been found, but it is

difficult to draw firm conclusions due to a lack of high-quality studies [10]. While several studies have investigated the efficacy of biofeedback for chronic back pain, the unique contribution of biofeedback to the treatment outcome remains unclear because it is often used in combination with other interventions, for example, as part of CBT [4]. In meta-analyses, biofeedback has often been viewed as one type of behavioral therapy and has been pooled with other treatment options such as relaxation [11].

To further assess the efficacy of biofeedback in the psychological treatment of chronic back pain, we performed a meta-analysis examining short-term and long-term effects of biofeedback on relevant pain-related outcomes [4]. To examine unique contributions of biofeedback, we analyzed studies that reported that at least 25% of total treatment time consisted of biofeedback. For uncontrolled pre-post/follow-up data, we found small-to-medium effect sizes both for the short term and the long term for all outcome variables, including pain intensity, disability, muscle tension, emotional functioning (e.g., depression) and cognitive functioning (e.g., self-efficacy). When biofeedback was tested against active control groups (e.g., physical therapy, CBT, relaxation training), we found only small controlled effect sizes for pain intensity, emotional functioning and cognitive functioning, but no effect for disability. Only the outcome variable 'reduction of muscle tension' showed a medium-sized effect size among controlled studies.

These results suggest that psychological treatments for chronic low back pain might lead to greater improvements on most core pain-related outcomes outlined by IMMPACT [12] when these treatments are enhanced with biofeedback elements. Furthermore, descriptive data indicated that clinically significant improvement (measured by the 33% plus criterion [13]) for pain intensity was more common at both post-treatment and follow-up in therapies with biofeedback elements. These results are in line with those of Flor and Birbaumer [14], who compared the efficacy of EMG-based biofeedback, CBT and medical care for patients suffering from chronic musculoskeletal pain and found that biofeedback was effective and also yielded the highest proportion of clinically significant improvements at both post-treatment and follow-up. They concluded that EMG biofeedback “may be a superior treatment for chronic

Biofeedback as a psychological treatment option for chronic back pain **COMMENTARY**

musculoskeletal pain.” However, Newton-John and colleagues [15] and Glombiewski and colleagues [16] did not find significant differences between CBT and CBT enhanced with EMG biofeedback. Lack of statistical power may have been a limitation of these two studies, as very large sample sizes are required to demonstrate differences between two similar, active treatments.

In summary, while the results of the recent meta-analysis suggest that treatments enhanced with biofeedback may be superior to other active treatments, further research on this topic is needed.

Mechanisms of change: does biofeedback for chronic low back pain work through physiological or psychological mechanisms?

Although biofeedback ostensibly focuses on changes in physical processes, for example, the reduction of muscle tension in the lower back, the main treatment targets may be psychological rather than physiological. Research in the field of headache and migraine has suggested that biofeedback is effective due to an increase in self-efficacy rather than reduction of muscular tension or due to a combination of both mechanisms [8,9]. However, in the treatment of chronic back pain, the mechanism of change for biofeedback has not been well studied, raising the question of whether self-efficacy is also the main mechanism of change for chronic back pain.

Chronic back pain patients often show higher psychophysiological reactivity, resulting in more sympathetic arousal and higher muscle tension compared with individuals without chronic back pain [17]. In the recently published meta-analysis on the efficacy of biofeedback for chronic back pain, the greatest effect sizes were found for EMG-based outcome measures, that is, the reduction of muscle tension, when considering studies with active control groups (Hedges’ $g = 0.7$) and when testing against waitlist control groups (Hedges’ $g = 0.8$) [4]. In comparison with EMG-based outcome measures, cognitive outcomes, including self-efficacy, showed small-to-medium effect sizes (Hedges’ $g = 0.3$ for active controls; 0.6 for waitlist control groups) for pre-post-data. As mentioned above, biofeedback is often used for training in relaxation techniques or to increase flexibility and range of motion through stretching exercises. These interventions may lead to lower muscle tension and thus

may result in positive effects on pain-related outcomes. However, these effects also occur after pure relaxation trainings or physiotherapeutic exercises, and the effects are enhanced when biofeedback is added. Thus, it seems overly simplistic to state that an increase in ability to relax, and thus, a decrease in tension, fully accounts for the therapeutic efficacy of biofeedback treatment. Furthermore, Kapitza and colleagues [18] demonstrated that respiratory biofeedback was effective for several pain-related outcomes even if the feedback was a placebo. As a result, it appears that a mechanism other than the reduction of muscle tension is responsible for the efficacy of biofeedback as a treatment for low back pain.

What are the limitations of biofeedback in the treatment of chronic back pain?

Although the literature suggests that biofeedback is an important psychological intervention for individuals suffering from low back pain, there are serious limitations that must be taken into account. First, a recent study found that biofeedback did not significantly reduce pain-related disability levels in chronic low back pain [4]. We believe that the reduction of pain-related disability is a more important goal in psychotherapeutic treatments than is the reduction of pain intensity. It is critical to utilize more effective treatments for highly disabled low back pain patients, such as CBT with graded activity elements or the more recently developed *in vivo* exposure treatment [19]. Thus, as promising and effective biofeedback may be as a psychological treatment option, it should be kept in mind that biofeedback does not appear to every core domain of chronic low back pain pathology.

In addition, it seems that there are obstacles to utilization of biofeedback as a psychological treatment option. The variety of modalities and treatment targets provides interesting treatment opportunities; on the other hand, these opportunities may have a deterring effect – that is, if clinicians feel overwhelmed by the variety and complexity of options. Second, biofeedback hardware and software are added expenses, and clinicians may rightly question the usefulness of such expenses especially against the background that common CBT interventions are effective as well, and the unique contribution of biofeedback as a standalone intervention is still unclear. On the other hand, perhaps because the intervention is rather technical but noninvasive, biofeedback is well accepted by patients suffering from low

“Interventions specifically addressing level of functioning, such as *in vivo* exposure or graded activity, should be the core elements of psychological treatment programs for chronic low back pain with high disability.”

COMMENTARY Sielski & Glombiewski

back pain even when their compliance with psychological pain treatments is low. From experience with chronic back pain patients who are skeptical of psychological treatments, we have the impression that biofeedback is valued as an interesting intervention and seems to help patients to understand the biopsychosocial mechanisms of persistent chronic low back pain.

Further research is needed on biofeedback as a psychological treatment option for chronic back pain. One important issue that was addressed in this article was the question about the primary mechanisms of actions. To this end, experimental studies on mediators for biofeedback are needed. With a greater understanding about the mechanisms of action, therapeutic interventions could be further developed and adapted to enhance treatment outcomes. Furthermore, effects of biofeedback on actual behavioral outcomes should be investigated. If range of motion or flexibility of movements increases, how does this affect behavioral outcomes in a real-life setting? Chronic back pain patients show high scores on disability measures and are impaired in a variety of daily life activities. Thus, examination of changes in actual daily life activities appears important. In addition, outcome

measures such as work absenteeism could provide more insights about the external validity of measures of therapeutic success.

In conclusion, there is evidence that biofeedback is an effective psychological intervention for patients suffering from chronic back pain. Patients' acceptance of biofeedback is high, and we encourage clinicians to use biofeedback more frequently to support CBT interventions. However, individuals with chronic low back pain and high levels of disability should not be treated with biofeedback only. Interventions specifically addressing level of functioning, such as *in vivo* exposure or graded activity, should be the core elements of psychological treatment programs for chronic low back pain with high disability.

Financial & competing interests disclosure

The authors have no relevant affiliations or financial involvement with any organization or entity with a financial interest in or financial conflict with the subject matter or materials discussed in the manuscript. This includes employment, consultancies, honoraria, stock ownership or options, expert testimony, grants or patents received or pending, or royalties.

No writing assistance was utilized in the production of this manuscript.

References

Papers of special note have been highlighted as:
• of interest

- Dolce JJ, Raczynski JM. Neuromuscular activity and electromyography in painful backs: psychological and biomechanical models in assessment and treatment. *Psychol. Bull.* 97(3), 502–520 (1985).
- Neblett R. Surface electromyographic (SEMG) biofeedback for chronic low back pain. *Healthcare* 4(2), 27 (2016).
- Schwartz NM, Schwartz MS. Definitions of biofeedback and applied psychophysiology. In: *Biofeedback: a Practitioner's Guide (3rd Edition)*. Schwartz MS, Andrasik F (Eds). Guilford Press, NY, USA, 27–42 (2003).
- Provides a good overview of definitions and the development of biofeedback, for example, different rationales in a comprehensible manner.
- Sielski R, Rief W, Glombiewski JA. Efficacy of biofeedback in chronic back pain: a meta-analysis. *Int. J. Behav. Med.* doi:10.1007/s12529-016-9572-9 (2016) (Epub ahead of print).
- Provides extensive and the most recent data on the efficacy of biofeedback in chronic back pain indicating biofeedback treatment to lead to improvements on several pain-related outcomes in short and long term.
- Magnusson ML, Chow DH, Diamandopoulos Z, Pope MH. Motor control learning in chronic low back pain. *Spine (Phila. Pa 1976)* 33(16), E532–E538 (2008).
- Gevirtz RN, Schwartz MS. The respiratory system in applied psychophysiology. In: *Biofeedback: a Practitioner's Guide (3rd Edition)*. Schwartz MS, Andrasik F (Eds). Guilford Press, NY, USA, 212–244 (2003).
- Neblett R, Mayer TG, Brede E, Gatchel RJ. Correcting abnormal flexion-relaxation in chronic lumbar pain: responsiveness to a new biofeedback training protocol. *Clin. J. Pain* 26(5), 403–409 (2010).
- Nestorciuc Y, Martin A. Efficacy of biofeedback for migraine: a meta-analysis. *Pain* 128(1–2), 111–127 (2007).
- Nestorciuc Y, Rief W, Martin A. Meta-analysis of biofeedback for tension-type headache: efficacy, specificity, and treatment moderators. *J. Consult. Clin. Psychol.* 76(3), 379–396 (2008).
- Glombiewski JA, Bernardy K, Häuser W. Efficacy of EMG- and EEG-biofeedback in fibromyalgia syndrome: a meta-analysis and a systematic review of randomized controlled trials. *Evid. Based Complement. Alternat. Med.* 2013, 962741 (2013).
- Hoffman BM, Papas RK, Chatkoff DK, Kerns RD. Meta-analysis of psychological interventions for chronic low back pain. *Health Psychol.* 26(1), 1–9 (2007).
- Turk DC, Dworkin RH, Allen RR *et al.* Core outcome domains for chronic pain clinical trials: IMMPACT recommendations. *Pain* 106(3), 337–345 (2003).
- Jensen MP, Chen C, Brugger AM. Interpretation of visual analog scale ratings and change scores: a reanalysis of two clinical trials of postoperative pain. *J. Pain* 4(7), 407–414 (2003).
- Flor H, Birbaumer N. Comparison of the efficacy of electromyographic biofeedback, cognitive-behavioral therapy, and conservative medical interventions in the treatment of chronic musculoskeletal pain. *J. Consult. Clin. Psychol.* 61(4), 653–658 (1993).
- Newton-John TR, Spence SH, Schotte D, Wing C, Mary S, Street P. Cognitive–

B: Tabellarischer Lebenslauf und Publikationen

[Der Lebenslauf ist nicht Teil der Veröffentlichung.]

Publikationen in Fachzeitschriften (Peer-Reviewed)

Sielski, R., Lucke, S., Uengoer, M., & Glombiewski, J.A. (submitted). Forming implicit and explicit attitudes toward movements: The role of evaluative conditioning in the acquisition of fear-avoidance beliefs.

Sielski, R., Glombiewski, J.A., Rief, W., Crombez, G., & Barke, A. (submitted). Cross-cultural adaptation of the German Pain Solutions Questionnaire: an instrument to measure assimilative and accommodative coping in response to chronic pain.

Sielski, R. & Glombiewski, J.A. (2016). Biofeedback as a psychological treatment option for chronic back pain. *Pain Management*. doi: 10.2217/pmt-2016-0040

Sielski, R., Rief, W., & Glombiewski, J.A. (2016). Efficacy of Biofeedback in Chronic back Pain: a Meta-Analysis. *International Journal for Behavioral Medicine*. doi:10.1007/s12529-016-9572-9

Bliemel, C., **Sielski, R.,** Doering, B., Dodel, R., Balzer-Geldsetzer, M., Ruchholtz, S., & Buecking, B. (2016). Pre-fracture quality of life predicts 1-year survival in elderly patients with hip fracture – development of a new scoring system. *Osteoporosis International*, 27, 1979-1987. doi:10.1007/s00198-015-3472-8

Ausgewählte Kongressbeiträge

Sielski, R., Lucke, S., Uengoer, M., Rief, W., & Glombiewski, J.A. (2016). Verändert Evaluatives Konditionieren nicht nur Überzeugungen, sondern auch Verhalten? Poster präsentiert beim 34. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie der DGPs, Bielefeld, Deutschland.

Sielski, R., Lucke, S., Uengoer, M., Rief, W., & Glombiewski, J.A. (2015). Acquisition of Fear-Avoidance Beliefs in an Experimental Setting. Vortrag präsentiert beim Pain Research Meeting. Genk, Belgium.

Sielski, R., Lucke, S., Uengoer, M., Rief, W., & Glombiewski, J.A. (2015). Entstehen Fear-Avoidance Beliefs bei chronischen Rückenschmerzen durch Evaluatives Konditionieren? Eine experimentelle Untersuchung. Poster präsentiert beim 9. Workshopkongresses für Klinische Psychologie und Psychotherapie & 33. Symposium der Fachgruppe Klinische Psychologie und Psychotherapie der DGPs. Dresden, Deutschland.

Sielski, R., Rief, W., & Glombiewski, J.A. (2014). What is the effect of evaluative conditioning on back-stressing movements? An experimental study. Poster präsentiert beim Pain Research Meeting, Maastricht, Niederlande.

Sielski, R., Rief, W., Glombiewski, J.A. (2013). Introducing a new paradigm in experimental pain research: can fear-avoidance beliefs be formed through evaluative conditioning? Poster präsentiert beim 43. Annual Congress der European Association for Behavioral and Cognitive Therapies, Marrakesch, Marokko.

C: Eidesstattliche Erklärung

Hiermit versichere ich, dass ich meine Dissertation

„Fear-Avoidance Beliefs, Coping und Biofeedback

Was wird bei der Entstehung und in der Behandlung chronischer Rückenschmerzen gelernt?“

selbstständig ohne unerlaubte Hilfe angefertigt und mich dabei keiner anderen als der von mir ausdrücklich bezeichneten Quellen und Hilfen bedient habe.

Die Dissertation wurde in der jetzigen oder einer ähnlichen Form noch bei keiner anderen Hochschule eingereicht und hat noch keinen sonstigen Prüfungszwecken gedient.

Marburg an der Lahn, Dezember 2016

Robert Sielski